JOURNAL of the AMERICAN VETERINARY MEDICAL ASSOCIATION

VOLUME XCVI, NUMBER 756 MARCH 1940

TABLE OF CONTENTS, PAGE II

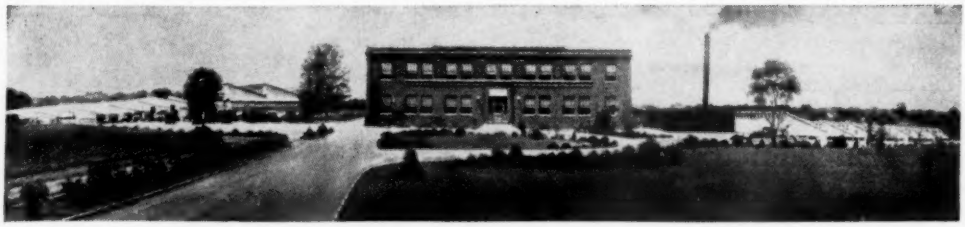
"CORN STATES"

and the

VETERINARIANS' OUTSTANDING PROBLEM

Reminding the general public of the debt it owes to farm animals is a praiseworthy undertaking, and

one of the most subtle problems facing the practitioners of veterinary medicine in the proper pursuit of their professional duties and legal rights.



Home Plant of The Corn States Serum Company

The increasingly high rating of veterinary science and the present confidence in its rational application were earned through the clinicians selected as competent to represent the capable producers of their supplies in the field of practice, but,

the dependence of mankind upon domestic animals and upon the skillful management they require to make their production a thriving industry would continue to remain unweighed but for the aid of educational programs calling attention to the wisdom of consulting only college-trained men to cope with disease.

THE VETERINARY SERVICE OF 1940 MAY BE PROUD OF ITS RATING

THE

CORN STATES SERUM COMPANY

OMAHA

Journal
of the
American Veterinary Medical Association

221 N. La Salle St., Chicago, Ill.

VOL. XCVI MARCH 1940 NO. 756

"Visit the National Capital in August." This is the slogan of the 77th annual meeting of the Association, to be held in Washington, D. C., August 26-30, 1940. And it is your cordial invitation to make plans now to be in attendance at this important session.

A Historical Prospectus of the Washington Session

THE FIRST and only annual convention of the Association ever held at the national capital was convened for a two-day session at Willard Hall, Willard Hotel, September 15, 1891.

Rush Shippen Huidekoper, whose name is indelibly inscribed in the archives of American veterinary medicine, was the Association's president, and W. Horace Hoskins, pioneer figure in organized veterinary activities, was the secretary. Both were reëlected, and both resided in Philadelphia, which at that time was sharing with New York City and Boston the distinction of being the center of veterinary development.

The report of the meeting—the 28th—gives the attendance as "about seventy-five." Sixty sat down at the banquet, which was a glamorous event outstanding in more ways than one ($5.00 for a banquet ticket, to cite an example). Secretary of Agriculture J. M. Rusk was the principal speaker and, incidentally, he was the first secretary of agriculture. His theme was "The mutual interests of agriculture and veterinary science," a subject which was to gather new importance with the passing years. The speech extolled the labors of the veterinary profession "when national wealth is threatened by epizoötic diseases."

C. C. Lyford of Minnesota read a paper entitled "Barren Mares," and W. L. Williams, then of Purdue University, talked on rachitis. With a few modifications these papers would be presentable today, or nearly a half century later (still sound on every scientific and literary count). Williams is the only living person who contributed to the program. There was a heated discussion between Williams and Schwartzkopf on the contagiosity of actinomycosis which grew out of the report of the special Committee on Meat Inspection. Such discussions were staged frequently during the period preceding the founding of meat inspection, and foreign governments were pointing critically to the lack of such inspection in our industrial scheme as a good and sufficient reason for barring our meat from export trade. Nothing more clearly illustrates the resourcefulness and ambitions of the A.V.M.A. than the reports, and discussions of the reports, of this special Committee on Meat Inspection, created at the 1890 meeting in Chicago. That these led to the founding of the marvelous meat-inspection service that the American people now enjoy is self-evident.

In his presidential address Huidekoper said, "I should like to see it become a requisite condition to qualify a man to become a member of the Association that he be a member in good standing in his county and state associations." Huidekoper thus foresaw what the Association is trying to do in 1940. He also advised that the jurisdiction be expanded in reality over the

General view of the principal buildings of the United States Department of Agriculture. The Administration building, which houses the chief administrative offices of the bureau of animal industry, is seen at the left.

whole country, and pointed to the large meeting in Chicago the year previous in testifying to the enthusiasm in organization developing in the Midwest.

Among the 25 successful applicants for membership at the Washington session who were to make history were W. B. Niles, M. E. Knowles, M. H. Reynolds, J. C. McNeil, N. S. Mayo, J. P. Turner, S. Brenton and J. T. Ryan. Three applicants were rejected as undesirable, and the secretary was directed to notify a number of specified members that they must remove objectionable material from their letterheads or forfeit their memberships.

The Washington meeting of 1891 was not large but its influence was vast. The controversies over the insufficiency and imperfections in the veterinary educational program, the need of a federal meat-inspection service, the problems of hog cholera, Texas fever, and contagious pleuropneumonia of cattle were interspersed prominently among the questions of current interest to practitioners, just as is done in the programs of the Association today—just as will be done when the Association again convenes at Washington, D. C., August 26-30, 1940.

LITERARY PROGRAM NOW BEING PLANNED; CONTRIBUTORS URGED TO COMMUNICATE PROMPTLY WITH SECTION OFFICERS

Members desiring to present papers at the forthcoming session are requested to communicate promptly with the secretary of the section to which their contribution belongs. Requests for a place on the program of the general sessions should be sent to the executive secretary, who acts as chairman of the Committee on Program. The other members of this committee are

the secretaries of the various sections. The membership of the Committee is as follows:

W. R. KRILL, *Section on General Practice*, College of Veterinary Medicine, The Ohio State University, Columbus, Ohio.

M. O. ROBINSON, *Section on Sanitary Science and Food Hygiene*, Alabama Polytechnic Institute, Auburn, Ala.

FRANK THORP, JR., *Section on Research*, Colorado State College, Fort Collins, Colo.

ROY E. NICHOLS, *Section on Small Animal Practice*, College of Veterinary Medicine, The Ohio State University, Columbus, Ohio.

C. A. BRANDLY, *Section on Poultry*, Regional Poultry Research Laboratory, East Lansing, Mich.

L. A. MERILLAT, *General Sessions*, 221 N. La Salle St., Chicago, Ill.

It is imperative that all papers be completed and in the hands of the executive secretary not later than July 15. An abstract of 200 to 500 words should accompany the manuscript. All of the abstracts and as many of the manuscripts as possible will be printed for distribution at the meeting. No unreasonable restrictions will be made on the length of papers, since the reporters will defend them rather than read them verbatim.

LOCAL COMMITTEE ON ARRANGEMENTS MEETS; TENTATIVE PROGRAM PLANNED

At its first definitely "down to business" meeting, the local Committee on Arrangements gathered at the Mayflower Hotel in

LOCAL COMMITTEE ON ARRANGEMENTS FOR THE 77TH ANNUAL MEETING

Front row, seated (left to right): Col. R. A. Kelser, banquet arrangements; Adolph Eichhorn, associate chairman; J. R. Mohler, chairman; I. M. Cashell, secretary; A. E. Wight, alumni dinners; W. M. Mohler (although not an official member of the Committee, Dr. Mohler is secretary of the District of Columbia Veterinary Medical Association, which will have a prominent part in the pre-convention planning).

Middle row (left to right): Mason Weadon, small animal clinic; Cassius Way, president of the A.V.M.A.; W. J. Hall, poultry and sheep clinic; Willard H. Wright, president's reception; L. A. Merillat, executive secretary of the A.V.M.A.; H. W. Schoening, educational exhibits.

Back row (left to right): Mark Welsh, publicity; L. T. Giltner, publicity; H. W. Jakeman, chairman of the Executive Board; J. F. Crosby, technical exhibits; J. P. Turner, large animal clinic. (Absentees G. W. Gillie, who is in charge of publicity, and H. M. O'Rear, who will supervise the general entertainment, are pictured separately on page 298.)

Washington, D. C., on Saturday, February 10, and drafted a tentative course for the convention. According to their report, the five-day event will proceed as follows:

Monday, August 26

Executive Board meeting and committee meetings.

7:30 P. M.—Meeting of House of Representatives.

Tuesday, August 27

10:00 A. M.—General session with opening ceremonies.

12:30 P. M.—Luncheon for the ladies and children in the Mayflower Hotel, followed by a meeting of the Women's Auxiliary in the Italian Gardens.

Afternoon. — General session. (Tentative speakers: Drs. Wyckoff, Meyer and Huddleson.) Card party for the ladies in the grand ballroom.

6:00 P. M.—Alumni dinners (to 9:00).

9:00 P. M.—President's reception and dance in the Pan-American Union building or in the grand ballroom of the Mayflower.

Wednesday, August 28

9:30 A. M.—Buses for the ladies to the dock for trip to Mount Vernon.

2:00 P. M.—Men will join ladies at Mount Vernon.

7:30 P. M.—Annual banquet and dance.

Thursday, August 29

9:30 A. M.—Bus trip for the ladies around the city. (A similar trip will be offered Friday morning.)

12:30 P. M.—Luncheon for the ladies in the Mayflower. Following, free tickets to moving picture theatres will be given to the ladies and children.

Afternoon.—Executive Board meeting. (This may be held late in the afternoon en route back on the boat from Mount Vernon.)

7:30 P. M.—Second meeting of the House of Representatives.

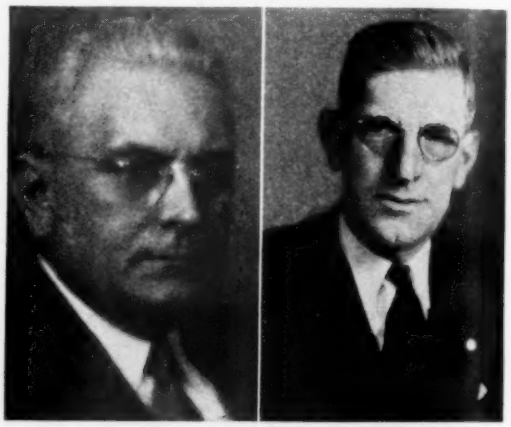
10:30 P. M.—Final general session.

Friday, August 30

All-day large and small animal clinic. (An optional trip will be offered to men interested in visiting the research laboratories at Beltsville, Md., and Fort Myer.

If possible, the entire convention will take Wednesday afternoon (the 28th) for the trip to Mount Vernon, which will occupy about four hours. As regards the annual banquet, it is thought advisable to have high class entertainment with a floor show, which would cost about $500. At the meeting of the local committee, it was recommended that some outstanding speaker be secured for the banquet, perhaps Senator Josh Lee of Oklahoma.

The possibility of holding part of the large animal clinic in the ballroom of the Mayflower and the remainder at Beltsville and Fort Myer was discussed at length by the Committee. Dr. Way, who traveled from New York City to attend the meeting, urged that the session in the ballroom be a demonstration clinic, or a dry clinic, with actual operations taking place only at Beltsville. From the standpoint of publicity, it was urged that the clinic in the ballroom consist of such features as blood transfusions, special anesthesia, the use of dextrose saline therapy and the restraint of large animals.



Important figures in the 77th convention set-up are H. M. O'Rear (left), who is in charge of entertainment, and Congressman George W. Gillie, who will direct publicity.

The publicity committee plans to obtain the names of veterinarians who will attend the convention and to mail stories to the home-city newspapers in order to publicize the part of individual members in the activities of the session. Contacts already have been made with Washington radio stations for broadcasts by prominent veterinarians during the meeting, and it is proposed to have a speaker discuss the value of veterinary service in wartime. A broadcast on the Farm and Home Hour and participation in the "Americans at Work" program, which is presented over the Columbia Broadcasting System, also are being arranged.

WASHINGTON'S HISTORICAL ENVIRONS AFFORD LUXURIOUS SIGHT-SEEING

Well-known, historical spots in the suburbs of Washington, D. C., are an out-

standing attraction and, in all probability, will in themselves interest extra numbers in attending the convention.

Fairfax county, Virginia, Bladensburg, Georgetown, Chevy Chase, Fort Myer, Fort Belvoir and the United States Military Academy at Annapolis, Md., are all within a short radius of the Capitol City.

The George Washington National Masonic Memorial on Shooters Hill, lying a short distance west of Alexandria, a suburb of Washington, symbolizes the beneficent influence of the life and character of George Washington on the government of the United States. Modeled after the ancient towers which were erected in harbors to serve as beacons to guide mariners safely to port, it is an unusual work of architecture.

Bull Run, scene of the historic battle of the Civil War, is a short drive from Washington. Bladensburg, now a quiet Maryland village some seven miles outside the Capitol City was, in 1814, taken and burned by the British. It is little changed in appearance since the battle day.

Veterinarians and their wives will find Dunbarton House in Georgetown an interesting place to visit. It is now a museum of colonial life in the United States. Key Mansion in Georgetown is the old home of Francis Scott Key, author of the "Star Spangled Banner." It has been remodeled and is now used for business purposes.

Fort Myer, the principal military post near Washington, was occupied by the military forces when Col. Robert E. Lee resigned from the army in 1861. Officers' quarters, barracks, drill hall, and hospital, on well-paved streets, make it a model army post.

Fort Belvoir, near Mount Vernon, is the home of the Engineer School and is headquarters for the Corps of Engineers' supplies and training. The garrison at Fort Washington on the Maryland side of the Potomac, near Mount Vernon, is used particularly in the summer season for C.M.T.C. training.

At the head of the Conduit Road on the Maryland side of the river, the Great Falls of the Potomac is certainly worthy of a visit. The water supply for Washington is obtained by a dam at that point.

Annapolis, the capital of Maryland and the home of the United States Naval Academy, is on the shore of Chesapeake Bay, and within easy driving distance of Washington. It is unique in its historic buildings dating from colonial times and the many interesting features of the naval academy activities. It is the only state capital in the country without a steam railroad connection.

PLAN NOW TO "VISIT THE NATIONAL CAPITAL IN AUGUST"

The five-day 77th annual meeting will be a prominent, colorful event that every veterinarian who is interested in the welfare of his profession ought to attend. A publicity machine primed for precision will focus the nation's eyes on the proceedings of this meeting and, without a doubt, the significance of the veterinary profession from the public view will attain an all-time high before the meeting comes to a close.

Organized Medicine Not Reactionary*

We quote: "While captious critics have been denouncing the American Medical Association as reactionary and obstructive, its members have been conducting numerous and extensive experiments during the last seven years in search of ways of organizing payments for medical service and adjusting the burden of medical costs to the abilities of the various economic classes. Never have so many, so varied or so significant projects been carried out in any other country."

Wilhelm Conrad Roentgen was born at Lennep in the German Rhineland in 1845 and died at Munich in 1923. The date of the discovery of the ray bearing his name is given as November 8, 1895. This famous physicist, neither rich nor poor, was a true scientist. He took no personal profit from his historic discovery.

*The Journal of the American Medical Association, cxiii, December 30, 1939, p. 2421.

So-Called Hemorrhagic Septicemia*

By W. A. AITKEN, D.V.M.
Merrill, Iowa

FEW DISEASES of domestic animals have been the subject of more controversy among veterinarians than hemorrhagic septicemia. Because of the wide distribution of the Pasteurella organism (= *P. boviseptica*) some observers choose to view it as only a secondary invader, whereas others believe that the bipolar bacillus, while usually of low virulence, can under certain conditions become very pathogenic.

Careful research by many eminent workers years ago determined the characteristic courses, symptoms and lesions of hemorrhagic septicemia in the susceptible species and established this disease as a definite entity.

Briefly, it was described as a sporadic but at times an enzoötic disease which could vary from a peracute infection, striking sudden death, to a chronic, lingering illness. The symptoms ranged from a simple fever with depression sometimes varied by mental disturbances, edematous swellings and hemorrhagic discharges to chronic unthriftiness, usually with pulmonary involvement. The characteristic lesions ranged from those of acute septicemia to chronic organic infections.

Most spectacular of the lesions were the small hemorrhages and lymph stagnations of the acute cases. The hemorrhages were scattered throughout the body, especially in the areolar tissues, serous membranes, heart and thymus gland; the lymph accumulations were noted in swollen lymph nodes, edematous tissues and, occasionally, as excessive fluid in the body cavities.

Various forms of the disease were generally accepted, including septicemic, pulmonary, intestinal, nervous and even cutaneous and arthritic forms.

Pasteurellosis was thought to occur naturally in several species of animals and it was believed that it could be produced experimentally in others. In fact, hemorrhagic septicemia as a disease seemed to be nearly omnipresent and omnipotent.

With this disease covering such a wide range, it was inevitable that many afflictions due to a variety of causes could not be differentiated from hemorrhagic septicemia in the field or even in the diagnostic laboratory, since the finding of the bipolar organism in the diseased tissues did not prove its etiological rôle.

It is small wonder that the pendulum swung too far and the popularity of hemorrhagic septicemia soon greatly exceeded its actual significance. It was eagerly accepted by the laity because its name is impressive, mysterious and intriguing. This, as well as the ease with which treatment could be applied, enhanced its popularity with the veterinary profession. They found it a ready alternate when a diagnosis was in doubt, a silent martyr when an alibi was needed and, in the case of the unscrupulous practitioner and quack, it was often a "gold mine." Now its popularity is waning; one by one, conditions formerly thought to be due to the Pasteurella organism are being found to be due to other causes.

Without a careful census it would be presumptious to attempt to judge the prevalence of pasteurellosis in all susceptible species in all localities. Therefore, the following remarks will be confined to the incidence of diseases which resemble hemorrhagic septicemia in the large farm animals, namely, cattle, sheep, horses and swine, as observed in the western part of the Corn Belt.

To hold that the Pasteurella organism is never the primary pathogen in a disease of these species would be an arbitrary position, but we suggest that when one diagnoses the ailments of these animals as hemorrhagic septicemia, he is probably in error. In other words, the majority of cases diagnosed as pasteurellosis will probably not yield the Pasteurella organism and, if they do, it

*Presented before the Section on General Practice at the 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1939.

probably is only a secondary invader. The writer's attitude will be vigorously opposed by many veterinarians, and in some cases perhaps rightfully so. No one is expected to concur without proof, but all who have been diagnosing hemorrhagic septicemia without laboratory confirmation should scrutinize their suspected cases critically and submit specimens from a few which they consider typical to a competent pathologist for bacteriological identification and interpretation.

In the absence of a laboratory diagnosis, and in some cases in contradiction to it, the practitioner often must judge the accuracy of his diagnosis by the success of the treatment applied. Such a gauge is quite reliable in some diseases, such as anthrax and swine erysipelas, where if the specific serum is administered in time, the benefit is conclusive. However, in the case of hemorrhagic septicemia there is sufficient experimental evidence of the inefficacy of specific biological products to prevent any apparent benefits of their use from being advanced as proofs that the disease treated was in reality hemorrhagic septicemia.

DISEASES ERRONEOUSLY DIAGNOSED IN CATTLE AS HEMORRHAGIC SEPTICEMIA

It would be impossible to list all the conditions which have been incorrectly diagnosed as hemorrhagic septicemia. However, a few conditions from our own experience which either were or could have been called hemorrhagic septicemia will be discussed.

Corn-Stalk Disease.—Our first major disillusionment was in so-called corn-stalk disease, which chiefly affects young cattle. Although it is at least a quarter of a century since some evidence indicated that this condition is due to a toxin, its confusion with hemorrhagic septicemia has existed because the clinical symptoms and lesions are quite typical of those credited to peracute cases. It occurs in the late fall and early winter, when it seems reasonable to suppose that changes in the feed and weather might lower the animals' resistance to the ever-present Pasteurella organism. Corn-stalk disease runs a rapid course, illness being noted usually in less than twelve hours, and it seems always to be fatal. Briefly, the symptoms are depression followed in some cases by mania, then coma and death. The temperature history is seldom available as the veterinarian sees few cases in the early stages, but actually the temperature, which is at first normal, soon drops and often is in the low 90's in the coma stage. Postmortem findings are chiefly 1) small hemorrhages throughout the areolar tissues, but most constantly on the heart and thymus; 2) edema of the gall bladder which sometimes extends along the duodenum; 3) cyanosis with some swelling of the liver in most cases; and 4) congestion of the mucous membrane of the abomasum and small intestine in some.

The constancy of the subnormal temperature, cyanotic liver and edematous gall bladder, and our inability to demonstrate the bipolar or any other organism at the laboratories, soon convinced us that this is not an infection. Then, vaccination, which gave splendid results as long as the cattle were kept out of the stalk fields, was often proved ineffective as soon as they were returned to the stalks. However, the difference between this condition and hemorrhagic septicemia was not defined until the fall of 1935 when, encouraged by the spectacular recoveries from the recently established use of intravenous medication in cases of hydrocyanic acid poisoning, we tried various solutions intravenously in these "corn-stalk" cases. To our great surprise, we soon succeeded in getting a cow apparently in the throes of death to improve to a point where she seemed practically normal. However, she suffered a relapse after a few hours and was soon again near death. The treatment was then repeated and she was again revived, but to a lesser degree, and soon she died. All we had accomplished was to prolong life about ten hours.

Further experimenting proved the beneficial agent to be dextrose, which when given intravenously seems temporarily to neutralize the toxin in the system. Many cases have since been revived in this way but, in spite of various supplementary treatments, a relapse always occurs and death has in each case been postponed only a few

hours. The only real value of this discovery to date is that it conclusively proves that this is not an infectious disease. While this disease is confined to the Corn Belt, other regions have reported other plant poisonings which could readily be confused with hemorrhagic septicemia.

Coccidiosis.—Another condition often called hemorrhagic septicemia in the past is coccidiosis. It frequently affects young cattle shortly after they have been shipped, although many times only home-raised cattle are affected. Undoubtedly, many mild cases go undetected, but severe cases often prove fatal in a few days. The hemorrhage in the feces suggests hemorrhagic septicemia of an intestinal form but the true cause can usually be demonstrated readily by a microscopic inspection of the fecal material or intestinal scrapings.

Coccidiosis in cattle does not seem to assume the enzoötic-like rôle that it does, for instance, in chickens; instead, it is quite sporadic. The reason for this, suggested by some parasitologists, is that clinical cases apparently result from lowered resistance of the host, which facilitates the invasion of the intestinal epithelial cells by the coccidia, which are apparently normal inhabitants of the intestine. As in any such sporadic conditions, preventive treatment will usually give phenomenal results regardless of the agent used. Therefore, some cattle feeders who have vaccinated for this intestinal form in the past will insist on having their cattle vaccinated whenever coccidiosis appears. Such vaccination could be justified only if the vaccine would help to prevent the phase of lowered resistance which seems to allow the invasion of the coccidia.

Deficiency Conditions.—Some deficiency conditions might, in isolated cases, be diagnosed as hemorrhagic septicemia. On several occasions, especially during recent drouth years, calves that appeared thrifty suddenly developed convulsions terminating in death. Autopsy occasionally reveals petechial hemorrhages on the heart, which might suggest a peracute hemorrhagic septicemia. Specimens from these cases were always sterile and, since calves usually recover from such convulsions, and in some cases from repeated recurrences, it is evident that this condition also is not infectious. Correcting the diet usually ends this trouble.

Sweet Clover Poisoning.—A peculiar condition which might have been diagnosed as hemorrhagic septicemia was seen on May 3, 1939. A client reported that he was losing his calves when they were 2 to 4 days old after a 6- to 24-hour illness. Labored breathing and weakness were the only symptoms noticed. No temperature history was available. The calves were born to Hereford cows confined in a dry lot, where their food consisted of grain and sweet clover hay only. Of the eleven calves born in the lot, four of the latest had died. An autopsy on two revealed only anemic tissues and rather extensive hemorrhages scattered throughout the subcutis, in the intercostal tissues, in the thoracic cavity and along the abdominal aorta.

Although it seemed improbable that so-called sweet clover poisoning could affect baby calves, either prenatally or postnatally, when the cows seemed normal, sweet clover poisoning was nevertheless suspected. The herd was immediately placed on pasture and the hay discontinued. Several more calves were born soon after this but no more losses occurred and specimens from the autopsied calves proved sterile. This may not have been due to the sweet clover hay but, certainly, it was an infection. In general, the magnitude of the hemorrhages with the dietary history should help identify sweet clover poisoning.

"Flu."—Another peculiar condition which suggested hemorrhagic septicemia was seen in February of 1939 in a herd of 20 closely stabled dairy cows. The owner had purchased a cow just shipped about 300 miles and stabled her with the other cows. On the second day she calved, on the third day she was very ill, and on the fourth day she died. From the owner's report, it seemed that this was a case of pneumonia. The second day after death an alarming condition existed in the herd. Half of the cows were ill, four being very depressed, trembling, with temperatures of 106 to 107° F., completely off feed and with a complete cessation of milk flow. Six others were no-

ticeably ill, with temperatures of 103 to 104° F. A few were coughing. Whether the treatment was logical and whether it in any way influenced the course of the disease is open to doubt, but each cow was given 10 cc. (2.5 dr.) of pulmonary mixed bacterin. All of the sick animals were given a dose of nux vomica and sulfanilamide, and sodium bicarbonate was placed in the drinking water. The next day the ten sick cows were again normal, but two other cows had developed high temperatures and were similarly treated. A day later all seemed normal. Since no autopsies could be performed, laboratory aid was not available, but, surely, such phenomenal recoveries could not have occurred in any extensive infection. Although we had never heard of such a diagnosis in cattle, we were tempted to call this "flu."

That vaccination with pulmonary mixed bacterin is not without dangers was demonstrated when one of these cows showed evidence of shock a few minutes after she was injected. It was again demonstrated a few days later, when a neighbor requested that his herd be similarly vaccinated. Three hours after the injection of the bacterin one yearling steer was found dead and another in a state of severe shock, but, fortunately, the latter recovered. Both seemed normal when vaccinated.

So-Called Shipping Fever of Cattle

The above-mentioned group of bovine maladies might readily be distinguished from pasteurellosis, but such is not the case with so-called shipping fever. A definite difference of opinion as to its etiology exists among practicing veterinarians, while the pathologists point to the frequent presence of the bipolar organism but usually doubt its primary rôle.

We have submitted only a few specimens from such cases to a laboratory, all of which failed to produce bipolar organisms. In October of 1938, a healthy Guernsey heifer was confined while calving in a pen recently occupied by cases of shipping-fever pneumonia. Two days later she was feverish and four days later she died of pneumonia. Lung specimens yielded diphtheroid organisms but even mouse inoculations failed to demonstrate any bipolar organisms. Whether or not shipping fever in cattle is ever pasteurellosis can be decided only by careful research.

Ovine Hemorrhagic Septicemia

As regards hemorrhagic septicemia in sheep, little experimental information is available. However, it seems that practitioners and pathologists alike now believe that most cases formerly diagnosed as hemorrhagic septicemia are in reality due to dietary or other disturbances. In 1934, due to the drouth, train loads of lambs and sheep were shipped into our territory to be fed on a contract basis. Most of the feeders were inexperienced and attempted to feed more sheep than their supply of satisfactory feed justified. The results often were tragic. One farm, for instance, took 1,000 lambs and lost 200 during one blizzardly night, and 330 in all over a period of three months. Many such cases were autopsied and no lesions were found to indicate anything but malnutrition. If lowered resistance is all that is required for the development of hemorrhagic septicemia, surely here the conditions for it were ideal.

We did find what we thought was hemorrhagic septicemia in lambs on September 30 of that same fall (1934). Three hundred and fifty-five lambs had been imported two weeks before and seemed healthy. Suddenly, several sickened and in two days eight died, none having seemed sick for more than 24 hours. Several were depressed, with temperatures around 105° F. Some showed nervous symptoms and several had edematous ears, nose and lips. One had pneumonia. Autopsy of four lambs revealed typical septicemic lesions in each. Therefore, the flock was vaccinated with hemorrhagic septicemia bacterin, each of the afflicted was given serum, an antiseptic was used in the drinking water, and they were all confined to a dry lot for a few days. Four of the sick died soon afterward but the balance made an uneventful recovery. Naturally, we were well pleased with the diagnosis and treatment until the report from specimens we had taken to the laboratory

proved negative. In the light of more recent information, this may have been an allergic condition or even a photosensitization, yet the scarcity of growing plants at that late date and the high temperatures of the sick lambs makes this seem doubtful. Just what was responsible for this condition is still not known.

EQUINE HEMORRHAGIC SEPTICEMIA

While equine hemorrhagic septicemia rarely if ever exists, a few capable practitioners are convinced that they do see cases. We have in eight years suspected it in only two cases, both of which proved negative at the laboratory. The most suspicious case was a gelding seen twice during his eight-hour illness. He was depressed, had a temperature of 104° F., was rather nervous and, at times, slightly colicky. Autopsy revealed many petechial hemorrhages on the serous membranes yet the laboratory found no microörganisms of significance.

PORCINE HEMORRHAGIC SEPTICEMIA OR SWINE PLAGUE

Swine plague, or hemorrhagic septicemia of swine, is still frequently listed as a current disease, without any qualifying limitations. This seems grossly misleading and unquestionably encourages erroneous diagnoses. Because of the difficulty in differentiating their various septicemic as well as chronic diseases, more swine specimens are submitted to diagnostic laboratories, in the Corn Belt, than all other large animals combined. Yet, at many of these laboratories the bipolar Pasteurella organism is practically never isolated or cultured from this large number of specimens. It may be found more frequently in other regions, but in the densely populated swine belt west of the Mississippi, it is very rare. One Iowa laboratory reports finding the Pasteurella organism in only one swine specimen since 1935, and that case later proved to be hog cholera. This is typical of the findings of most of the midwestern laboratories.

Such was not the case a few years ago, when the Pasteurella organism was frequently isolated from swine specimens. Significantly, there has been no parallel decrease in the number of septicemic cases in swine, which indicates that the bipolar organism probably never has been an etiological factor. There are many swine autopsies which present the lesions credited to hemorrhagic septicemia, but it is always other organisms, if any, which are isolated from these. Usually, it is the suipestifer or paratyphoid organism, but occasionally it is the erysipelas organism. Rarely, a streptococcus or diplococcus is found.

Fifty years ago the term swine plague clothed a robust disease, but with the differentiating first of hog cholera and since of many less specific ailments, the term now appears ridiculously like a ponderous shroud covering only a cadaverous remnant. We hope it may soon be restricted, revised or abandoned.

CONCLUSIONS

The practicing veterinarian seldom feels justified in making statements as emphatic as these, but we humbly do so with a conviction supported by the diagnostic aid of several well-qualified pathologists. We are favored by having one diagnostic laboratory just 20 miles from our office with excellent mail and highway connections, and we thus have been able to submit fresh specimens or living animals for diagnosis with little difficulty. In eight years we have failed, with one possible exception, to have the Pasteurella organism identified in a significant rôle in any specimens. We are confident that if other practitioners had such convenient aid, their experiences would in most cases be similar.

We would, therefore, sincerely suggest for the veterinary profession this slogan: "When You Call It Hemorrhagic Septicemia, Bring an Affidavit."

"Fresh air" means air free of particulate material; of 68 to 72° F.; humidity, 65 per cent; moving at the rate of 1 to 3 miles per hour; and exerting a pressure at sea level of 15 pounds per square inch. In other words, "fresh air" is a rare commodity.

Worm-Host Systems as Labile Mechanisms: A View of the Nematode-Ruminant Problem*

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SEVERAL years ago in China, while investigating hookworm, we were surprised, upon examining certain hospital records, at the number of cases of ancylostomiasis (=hookworm disease) which were recorded as primary or secondary diagnoses. As these cases occurred in an area where, according to our studies, the disease should not have appeared so widespread, further inquiry was made of the laboratory records and the house staff was questioned. We discovered that whenever no other clear-cut pathognomonic diagnosis presented itself, and hookworm eggs were present in the feces, the primary diagnosis automatically became ancylostomiasis. And, in all other cases where the laboratory report was positive, ancylostomiasis became the secondary diagnosis.

This is a good illustration, I think, of a widely prevalent point of view. The laboratory finds worm eggs, *ipso facto* the patient has a disease labeled by the name of the worm egg found: Uncinariasis, bunostomiasis, trichuriasis, a s c a r i a s i s, taeniasis, fascioliasis, haemonchosis, trichostrongylosis, etc. And, logically, why shouldn't it be true? After all, parasitic worms are large, able-bodied pathogens which have marvelous mouthparts or other holdfast organs, are strongly muscular and, among the nematodes particularly, have resistant cuticular protection. Moreover, many of them wander about the inside of the host corpus to an extent which not alone excites admiration for their Richard Halliburton tendencies but which still further emphasizes how much at their mercy they appear to hold the host. Wherever they penetrate, they soil their happy home by opening new avenues for the penetration of other smaller disease agents, they foul their environment within the host with their own fluid and non-fluid excreta, and erode room for themselves at the expense of host tissue. Not long ago I saw an autopsy of a mink, not much larger than an overgrown rat, from whose right kidney were removed three adult female *Dioctophyme renale*, each as large as an adult ascaris. The kidney had been reduced to a shell.

We all shudder. That is the classical subjective reaction which worm parasitism should produce in the observer. Unless you have studied helminthology in the very recent past, it is the only one you may have.

Despite, however, this classical idea that a host has as little chance against its chewing, sucking, poisoning, and eroding worm parasites as Cleopatra had against the asp, there is another story to tell and to consider. Theobald Smith used to emphasize that parasitism in its essence represents a host-parasite balance. If, as, and when the parasite is too destructive, it jeopardizes the life line by which it reaches from one generation to another. This philosophic view turns out to have adequate basis in fact even among our most horrendous parasites.

Within the past several years we have learned, by observation and experimentation, that hosts may react against worm parasites to the destruction of the parasites, as well as *vice versa*; that effective immunities may develop in parasitized hosts which not only cause the expulsion of resident infections but prevent all or most of further parasitism; that, in broad fundamentals, hosts react to worm parasites in the same way that they react to bacteria and viruses. Thus, worm-host systems classify as labile mechanisms, as biological systems "characterized by adaptability to change or modifi-

*From the Rockefeller Institute for Medical Research; presented at the 56th annual meeting of the Veterinary Medical Association of New Jersey, Newark, N. J., January 17, 1940.

cation." There is, then, a tide of battle, and it may go either way. It is not all *Blitzkrieg* by the parasites in host territory.

Over a period of years I have been studying something of this story as regards sheep, goats, and cattle in relation to their characteristic gastrointestinal nematodes. It was thus I was persuaded it would be of interest to you to present briefly some of the now known facts, as judged by my own work and that of others on the same or comparative problems.

What are some of these facts?

1) *Haemonchus contortus* is the blood-sucking, twisted wireworm of the ruminant abomasum, and by common report the most destructive single worm species in the ruminant problem. It is possible to show that the sheep host will develop an excellent immunity against this form, by just being fed more and more worms. For a while the host accepts the parasites in ratio, and they may build up in numbers to a point where measurable damage is done, with consequent retardation or loss of weight, depressed hemoglobin and unthrifty appearance. Then, a physiological crisis occurs, the infection is literally discharged in whole or in large part, and new worms fail to establish in the same ratios as earlier. The crisis may be marked by scouring. With the development of the immunity, weight and hemoglobin increase, unthriftiness disappears. The same result occurs on pasture, either with pure Haemonchus or with mixed infestations in which Haemonchus may be predominant.

2) The same phenomenon has been demonstrated in ruminants for *Ostertagia circumcincta* and *Trichostrongylus axei* by ourselves, for Nematodirus by Taylor in England, for Oesophagostomum and Bunostomum by Mayhew in Louisiana, and others. Beyond the ruminants, immunities are being demonstrated constantly. The first assumption now is that they will develop, rather than just the reverse, as was true a few years ago.

3) These immunities, while serviceable to protect the host, are usually not so-called sterilizing immunities; that is, a few worms at the subclinical level may be left in the host. Deparasitization can be shown on occasion to be greater in degree, however, than Ivory soap is pure, this being advertised at only $99^{44}/_{100}$ per cent. The fact that some worms remain is an additional reason why the presence of functional immunities against parasitic worms existed so long unrecognized. Unrecognized because just as in the hospitals in China, the demonstration of even one hookworm egg meant hookworm disease, the observer failing to take into account that the amount of infection present may have represented a small residuum of a vast amount of past exposure, rather than an accounting of all past exposure.

It may even be that all immunities which maintain themselves at a functional level are so maintained by residuals of infective agents.

If we can not demonstrate the presence of silent foci of viruses or bacteria, we must caution ourselves to remember that failure to demonstrate may be due to technical inability rather than to biological absence of microbic agents. On the other hand, the parasitic worms, being larger, can not escape observation when present in small amount.

4) These immunities are specific, not nonspecific. Our New Jersey pastures may have a dozen or a dozen and a half worm-parasite species maintained in them. If the immunity is specific, how is a host protected against them all? There are probably two answers to this. For one thing, not all worm parasites survive equally well in their external phases on pasture; climatological factors oppress them differentially; the fact that they are present by no means implies that they are all present at damaging levels. For a second point, usually some species predominates. Frequently it is Haemonchus. The host, in effect, then deals with the predominating form. If it becomes immunized to this predominating form, it can usually deal with the others in whatever order they arise.

Let me illustrate. We have at the Institute a field fenced for a herd of cattle which

is maintained exclusively by breeding within the herd. Early after the establishment of the herd, several helminth-free lambs were placed with the calves which were used to initiate the breeding stock. Subsequent examination of the sheep and cattle indicated that several nematode species had been present in these calves, among them Haemonchus. During three years this continued, and the sheep were then all removed. Following their removal, and without any treatment intervention whatever at any time, the combination of the immunity developed against Haemonchus by the cattle, plus the poor ability of Haemonchus to withstand the rigors of New Jersey winter conditions, caused this worm species to be completely eliminated from the area. It no longer shows at autopsies of calves, nor did it appear when new helminth-free sheep were reintroduced to test for its presence. In this case Haemonchus was probably the predominant infection while sheep were present to maintain it.

Another illustration. A young ram had been immunized by successive doses of Haemonchus administered by the experimenter. Following this he survived, without ill effects, pasture exposure to a small field upon which a pure Haemonchus infestation built up so high as to kill another animal. The third year the ram was placed in a pasture containing numerous nematode species. In midseason it was necessary to withdraw him indoors for a period of about two months. Just before withdrawal from pasture it was noticed that he was developing a rather heavy infection with what may have been Cooperia. This he maintained indoors. But, when reintroduced to the same heavily infested pasture, he promptly discharged his infection. Presumably he had not secured enough of the second species to cause immunization before withdrawal, but the process was completed after reinfection was reëstablished.

5) But you are saying to yourselves, "Aha, the worms do get away from him and damage and kill even *his* animals sometimes." Quite so. New knowledge does not necessarily supplant old knowledge; it rather supplements and modifies it, rounds out the picture. The gastrointestinal nematodes of ruminants are still pathogenic despite the fact that demonstrable, serviceable immunities develop against them. Well, then, under what conditions does this labile mechanism, which can be shown to favor the host, get out of hand and favor the worms?

I want now to try to give a part of that picture, as far as it has developed, but these studies, while a decade old, do not yet pretend to furnish all the answers.

1) For one thing the host mechanism is favored if the intake of worms is not too rapid or of overwhelming size at first. That is why young animals introduced into old stock pastures get in trouble before they can protect themselves. The old pasture infestation may be at a level which maintains immunity for the older members of the flock or herd, but that level may represent overdosing of infection to young stock. The same result may occur where animals have been roaming temporary, lightly infested pasturage and then are brought close to the building into old corrals, in which soil and forage conditions are such that they ingest all the scrubby pasturage and the concentrated external infestation which the enclosure permits.

2) For another thing, the host mechanism is favored if the host is well fed. Why is it that worm damage is so frequently reported in dry seasons? As Taylor points out, these are exactly the conditions under which the nutritive demands of growing or breeding animals are poorly met, and where the sheep, goats, or cattle with unsatisfied appetites forage right down to the grass roots. They thus evidence a food deficit. In trying to make it up they expose themselves to an inordinate amount of the worm infestation with which, in Hall's phrase, they soil their own table. Essentially, of course, this is overstocking, and it may occur in any season.

3) A third item. During periods when there are excessive constitutional drains on the vitality of individual animals, the immunity may be broken through. Such instances occur particularly in relation to breeding. While pregnancy ordinarily does

not interfere, occasional ewes will manifest reduced resistance late in the period. Lactation even more strikingly will permit the worms to gain an advantage, especially when ewes have suckling twins upon them. One of our most interesting curves is that of a ram who showed a strikingly lowered resistance to his worms during the month he was breeding the flock. Being continued on reinfestive pasture, he immediately and dramatically discharged his increased infection when breeding was over, the breeding dates having been back-checked after the lambs were dropped.

There are other types of excessive constitutional drains, such as severe chilling, following hard or prolonged storms; psychic and physical injury to flocks by dogs; etc. Dr. Smith has said that there is no kind of immunity which can not be broken through, and this naturally applies to immunities against the worms as well as others.

The picture is obviously not yet complete. We do not know, for instance, whether certain breeds or strains may not stand up against their worms better than other breeds or strains. While the demonstration of poor nutrition in causing a break-through of the immunity is clearly established both epizoötiologically and experimentally, we do not know on the positive side whether there may not be specific nutritive factors which are particularly valuable to the host in this respect. This pertains not only to such items as protein ratios and vitamins but also to inorganic constituents, all of which we are inclined to treat blithely in our ignorance.

Especially have we been handicapped in the absence of a good *in vitro* test for following more precisely the immune state of the host. Such tests we appear to be on the verge of acquiring, and it is possible that in a few years a much more precise accounting of the nematode-ruminant problem can be made.

You will realize, I am sure, that no attempt has been made to be exhaustive in this report. All that has been attempted is to give you an inkling of a widespread biological reaction with which Mother Nature has been taking care of her own against the ravages of animal parasites. To the reality of the reaction we can now bear witness, although its domestication, so to speak, in order that short-cut methods of immunization can be placed in your hands, is still in the future.

An Important Court Decision

Even though present at the time and helping, the owner of a mare is not responsible for accidents that befall the mare after she is turned over to the stallioneer to be bred. Once the operation starts, all of the responsibility rests upon the stallioneer, according to a court decision recently handed down in a French civil tribunal. The court further pointed out that the decision applies also to a horseshoer. In short, when a horse is turned over to a stallioneer to be bred or to a horseshoer to be shod, the owner is not its guardian. The entire responsibility of the operation rests with the stallioneer or the horseshoer, as the case may be.

The decision grew out of a suit for damages wherein the owner of a mare about to be bred was kicked by the stallion. The stallioneer held that the owner of the mare assumed equal risks with him in aiding in the execution of the performance, but the court did not agree and handed down the decision in favor of the injured man.

Some years ago in Chicago, a horse tied in a string of horses in a blacksmith shop waiting to be shod sustained a fractured leg when kicked by an adjacent horse. Both the horseshoer and the owner of the kicking horse were sued by the owner of the injured horse. The horseshoer alone was held responsible by the court on precisely the same ground set down by the French judge.

The greatest discovery of all times is the wheel. The backwardness of Inca and Mayan civilizations in the Western Hemisphere was due to the absence of domestic animals and wheeled vehicles. The one begot the other in Asia, where the two made possible the development of the more highly civilized populations.

Artificial Insemination of Dairy Cattle in Private Practice*

By ALFRED KISSILEFF, V.M.D.
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MUCH has been written, in the lay press especially, about artificial insemination and the part it will play in future livestock breeding operations. Cattle breeders have taken an active interest in it, but their enthusiasm is not shared in the same degree by the veterinary profession. Leading veterinary journals[1, 2] have recently sounded editorial warnings to practitioners to exercise caution in recommending artificial insemination until more detailed records are available. Williams[3] quotes several examples of failure to accomplish anything worth while by the interception of natural breeding processes.

On the other hand, the rapidly accumulating reports on artificial insemination indicate that there is a demand for it. There is, as yet, no conclusive evidence that artificial insemination is detrimental to the health or vigor of live stock, but it also remains to be proved precisely what ultimate benefits may be achieved. The faithful reporting of results, failures as well as successes, will in time enable us to evaluate the practice.

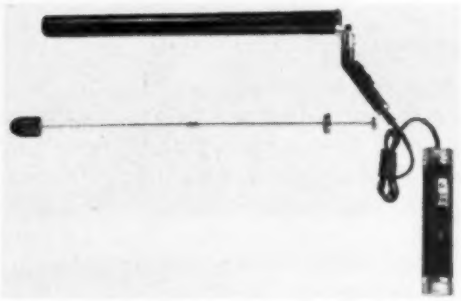
The material in this report is derived entirely from case records in private practice, the records covering all inseminations made during the period dating from July 1, 1938, to November 1, 1939.

TECHNIC

The technic used was essentially the same for each insemination. Semen was collected by means of the artificial vagina and stored, undiluted, in a thermos jar containing several ice cubes to keep the sample cool. Samples over 30 hours old were discarded. Dilution of the semen, when necessary, was made directly in the inseminating syringe, but, wherever possible, undiluted semen was used. Insemination was done through an illuminated vaginal speculum, 1.5 cc. (22 minims) of semen being deposited in the cervical canal with a capillary glass tube attached to a 2-cc. (30-minim) glass syringe.

The bulls used were all obtained from certified herds and were of good breeding. The number of times the bulls were used depended upon the number of cows to be bred. One bull has been used as many as 15 times in one month without affecting his percentage of conceptions.

In cases where the sample of semen was scanty, the bull was permitted to serve the artificial vagina more than one time. It was found that the sperms in a heavy sample of undiluted semen retained their vitality for a longer period than the sperms in a small sample, or one which was diluted. In connection with this finding, it is interesting to note that the degree of motility of the spermatozoa in stored semen is not necessarily a reliable guide to its ability to fertilize an ovum. Apparently, nonmotile sperms are capable of renewed activity when properly stimulated, and it is believed that the sperms are much more active in the genital tract of the cow than they are in a sample of stored semen. The addition of a drop of blood serum to semen several days old and showing feeble motility will immediately bring about intense activity even among the sperms which appeared to be entirely lifeless. This is, in some measure, reliable evidence that the motility of stored



Speculum used in the work.

*Presented at a meeting of the Keystone Veterinary Medical Association, Philadelphia, Pa., December 20, 1939.

spermatozoa is merely suppressed, and does not indicate the activity it may reach in the genital tract of the female.

Most of the cows listed in table I were inseminated as soon as possible after the semen was collected. There were, however, 30 inseminations made with semen over 20 hours old. Fourteen of these inseminations resulted in pregnancies and, up to the time of this writing, six of these pregnancies have terminated with normal calves.

The stage of the heat period has been said to exert some influence on the percentage of conceptions, it being claimed that cows bred at late heat are more apt to conceive.[4] Efforts to inseminate all of the cows at late or at full heat were made, and in no case was a cow inseminated at the first signs of heat.

Reasons for Resorting to Artificial Insemination

In order to understand the various reasons advanced in favor of artificial insemination, it is necessary for one to take into consideration the individual problems of breeding on different farms. On commercial dairy farms these are generally temporary difficulties. The appeal of artificial insemination to small herd owners is usually the same in all cases and is a permanent breeding problem, not a temporary one. The owner of a small purebred herd, or simply a purebred family cow, always encounters difficulty in getting his cow or cows bred to a good purebred bull. Most commercial breeders are reluctant to breed cows from outside sources because of the danger of introducing disease. The small breeder, therefore, must look elsewhere for a bull, and in many cases he uses any bull available as long as he can get the cow in calf. The small breeders, as a whole, welcome artificial insemination because it solves a problem which heretofore has always been more or less of a nuisance.

Included in the tabulation of results (table I) are 64 head which were all either family cows or cows in small purebred herds. Many of these cows are now in calf the second time to artificial service, and the owners are all satisfied to have their cows bred in this manner.

The advantages gained are numerous, and for the small herd owner there is no disadvantage that is at this time worthy of consideration. The possible damage to future generations which may be in the minds of the extremely cautious is not yet in evidence.

On commercial dairy farms the interest in artificial insemination is divided. Many breeders claim that it will hurt the sale of young bulls. There appears to be some justification for this contention, at least in the experience of the writer. Eight of the 25 small noncommercial farms listed in table I formerly owned herd bulls, but after contracting for artificial insemination, sold the bulls. All except one went for beef and it does not seem likely that these bulls will be replaced in the very near future. There are also some breeders who contend that their investments in buying and maintaining bulls are depreciated when a neighbor who, for example, owns but a few cows can have these cows bred to a good bull without going to the same expense. Despite this opposition by larger breeders, these same farmers are having occasion to use artificial insemination to good advantage.

Up to the time of this writing, 13 commercial dairy farms in the vicinity have requested artificial breeding and 109 cows have been inseminated. Each farm had a different reason for not breeding cows naturally. The farms are listed here as individual case reports in order to demonstrate the wide range of usefulness which artificial insemination may cover.

Farm 1.—A purebred herd of Guernseys were being raised on this farm. The owner of the herd had in the course of several years sold a number of cows for family use and had arranged to breed them as part of the sales agreement. In his contract for Bang's disease control he found that certain provisions prohibited the breeding of cows from outside sources. It was arranged to breed these cows artificially, and the practice was begun July 1, 1938. As the demand for outside breeding increased, he decided to breed his entire herd artificially and extend the use of the collected semen. Under this plan a total of 48 cows in his herd have been inseminated artificially. Seventy-six

inseminations were required to impregnate 41 cows, or 1.85 services for each pregnancy. Thirty-one conceived to the first service, eight to the second, and two to the third. Five were rebred naturally after one or more artificial services and two cows failed to conceive and were later slaughtered; a total of 15 inseminations were made on the two cows which were slaughtered. The reasons for non-conception in these two cows were never determined. Both had retained placentas at the last calving and it is likely that sterility resulted from some lesion suffered at that time. However, the lesions were never detected during life. At the slaughterhouse the genital organs were examined and nothing abnormal could be found.

Dr. Kissileff (left) demonstrates his method of artificial insemination.

The calves born to artificial service on this farm have been normal in comparison with calves born by natural service. The oldest calves are now 7 months of age and do not appear to be in any way different from natural service. The net results, as far as the relationship of services to the number of calves born, compare favorably with the breeding records of the two previous years in the same herd. There were two early abortions and one mummified fetus, for which no explanation can be given, since all were negative to Bang's disease and no other infection has been diagnosed.

It is the opinion of the writer that the method of impregnating had nothing to do with the incomplete gestation, since the same thing has happened in the same herd with natural breeding. This opinion, however, is open to challenge, in view of the fact that the cause of these conditions has not been thoroughly investigated.

The practice of artificial insemination in this herd has gained for the owner certain advantages which he considers valuable. He runs no risk of introducing any infection to his bulls by herd additions and he can safely breed a large number of outside cows to a young bull, which will enable him to determine the bull's prepotency at an earlier date than would otherwise be possible.

Farm 2.—The regular breeding schedule on this farm was interrupted by an unusual accident which resulted in temporary impotency of the herd sire. Briefly, the accident occurred as follows: On April 14, 1939, the bull was permitted to serve a cow which had come into violent heat six months after a previous breeding. Shortly after the service the cow delivered a mummified fetus. Thereafter, the bull was unable to breed a cow properly. When led to a cow in heat, he would mount and begin the act of service but would fail to bring it to completion. Two months later, the owner requested that several cows be bred artificially to one of the bulls at farm 1. His own bull was examined at that time and it was found that his penis was irregularly scarred on the glans and ventral part of the body. Samples of his semen collected by rectal manipulation showed active sperms, but since the bull could not ejaculate voluntarily, he was useless for service. It was the opinion of the owner that the bull was worth keeping provided that he would recover. The bull was given a complete rest from all sexual activity for three months, after which he was again used. Since September 1, he has been in active service. He is apparently recovered but is still unable to complete the service on any cow which is a little higher in the rear than usual.

TABLE I—Results of artificial insemination on 38 farms.

Farm No.	No. of Cows Inseminated	No. of Conceptions	No. of Inseminations Required	No. of Abortions	No. of Mummies	Slaughtered	Did Not Conceive to Artificial Service	Rebred Naturally and Conc.	Did Not Conceive to Either Kind of Service
1C*	48	41	76	2	1	2	7	5	2
2C	7	2	10	0	0	0	5	2	3
3C	5	5	7	0	0	1	0	0	0
4C	11	9	15	0	0	2	2	0	2
5C	10	9	12	6	0	6	1	0	0
6C	2	2	7	1	0	2	0	0	0
7C	5	5	8	0	0	0	0	0	0
8C	6	6	8	1	0	1	0	0	0
9NC†	14	13	20	0	0	1	1	0	0
10NC	3	3	3						
11C	7	3	15	1	0	0	4	4	0
12NC	3	3	3						
13C	2	0	2			2	2		2
14NC	1	1	1						
15NC	6	6	7		1				
16NC	1	1	2						
17NC	2	2	2						
18NC	3	3	5						
19NC	4	3	5			1	1		
20NC	2	2	4						
21NC	1	1	1						
22NC	3	2	5	1			1		
23C	1	0	3			1	1		1
24NC	2	2	3						
25NC	5	5	8						
26C	1	1	1						
27NC	1	0	3				1		1
28C	1	1	1						
29NC	3	3	4						
30NC	2	2	2						
31NC	1	1	2						
32NC	4	4	8						
33NC	1	1	2						
34NC	1	1	1						
35NC	1	1	2						
36NC	2	2	2						
37NC	1	1	1						
38NC	1	1	1						
Totals	174	148	262	12	2	19	26	11	11

*Commercial breeder. †Noncommercial breeder.

During the time this bull was retired, an attempt was made to maintain the breeding schedule by artificial insemination. The table shows that only two of the seven cows inseminated conceived. Two more were rebred naturally and conceived, and the other three are still open. It is worth mentioning that the two cows which conceived to artificial service both required but one service each. The two which later conceived naturally also conceived with but one natural service each. The remaining three were inseminated artificially a total of six times and also have been served regularly by the bull since September 1, but are not yet with calf.

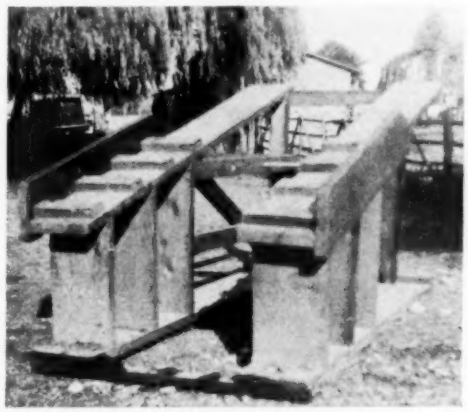
Artificial insemination was discontinued in the herd early in September, when the injured bull was returned to service. When it was found, however, that there were certain cows in the herd which did not conceive naturally, the owner again contracted to breed these difficult cows artificially. For some reason, this breeder (as well as several other breeders) believes that sterility can be overcome by breeding artificially, and is offering such subjects for insemination. The results are, of course, disappointing, for it

is unreasonable to expect conception to be accomplished artificially in a cow which does not conceive naturally. It is true that there are rare exceptions, but where the cow is at fault, the manner of service is of slight consequence.

Farm 3.—The owner of this farm had purchased five young granddaughters of the senior herd sire at farm 1. He desired to line breed these heifers back to the old bull, but because of the presence of Bang's disease in his herd this could not be permitted with natural service. The five animals were inseminated artificially, requiring a total of seven inseminations. Four conceived to the first service and one required three services. The latter animal had suffered a gaping wound of the vagina the day of the first insemination and it is thought that the persistence of this wound was the cause of temporary non-conception. Four of the five heifers bore full-term calves and one was slaughtered as a reactor three months after insemination. Artificial insemination was discontinued in the herd after January 1939, since the desired results were attained.

Farm 4.—This farm maintained a herd of 135 females and four males. Although the herd has been certified for Bang's disease for a number of years, there was a considerable amount of sterility and frequent abortions. A study of the breeding records and a careful examination of every breeding animal was made. After three months of work, it was found that 56 of the females and all four bulls were infected with trichomoniasis. A clean bull was obtained to breed the virgin heifers, and the cows which were known to have been previously infected were bred back to the infected bulls. The cows which had been bred to an infected bull but which were not known to be infected were classified as doubtful and, since it was not considered safe to breed them either to the clean bull or to the infected bulls, they are being artificially inseminated to the clean bull. Nine of these have been inseminated thus far and all have conceived. Two others, previously infected, also were inseminated at the owner's request, but failed to conceive. These two cows had been sterile for over two years and were subsequently slaughtered.

The control of trichomoniasis in this herd is a difficult problem, as can be seen. The original plan offered to the owner for control was to send all four infected bulls to slaughter and breed the entire herd artificially to a clean bull. The owner objected to this plan because it could not be determined when it would be safe to go back to natural breeding. Under the present system the disease is prevented from further spread but it will certainly be a very long time before the herd will be rid of it. In the meantime, as the infected cows are eventually found to be with calf, they are sold.



Breeding stocks used in artificial insemination work.

Whether the animals which are sold can become a source of infection to other herds is a matter for speculation—a matter which does not come within the scope of this report. It is, however, a subject which demands attention.

The use of artificial insemination in this herd is accomplishing its purpose and is an effective means of preventing the infection in the clean bull.

Farm 5.—The herd sire on this farm was suspected of transmitting Bang's disease, although his blood was negative. Following a severe outbreak during which all but one of the cows he bred aborted, it was decided to breed artificially to an outside bull until it could be determined from what source the infection was coming. The table

shows that the rate of conception in this herd was high but the control of the disease was not in any way made more effective. Six of the nine cows which conceived aborted in five to seven months. Three had full-term calves and are still negative animals. It can not be said in this case that artificial insemination was of value in controlling Bang's disease but it must be considered that the herd was already infected and any attempt to control it was not altogether wasted. The owner of the farm has discontinued all breeding until the herd can be cleaned up.

Farm 6.—This farm consisted of a large Jersey herd and two purebred Guernsey heifers which had been raised by one of the boys on the farm. The herd was infected with Bang's disease and outside service for the two Guernseys could not be obtained.

Both animals conceived to artificial insemination, but one aborted, was rebred and conceived again, and following a blood test of the entire herd, both of the animals reacted and were slaughtered.

Farm 7.—The loss of the herd sire on this farm created a problem which was solved with favorable results through artificial insemination. The bull at this farm developed lump-jaw and had to be slaughtered before one of his sons, the future herd sire, was old enough for service. During the interval, five of the cows were artificially bred to a bull on farm 1. The expense of buying a bull to breed only five cows was avoided and the normal breeding schedule was not delayed.

Farm 8.—A small commercial herd was being raised on this farm. The owner had but one bull, which was the sire of six females of breeding age. Another bull was owned by the same farm but he had been leased out for a year and, until he was returned, it was necessary to obtain service of an outside bull to avoid inbreeding. The six animals were artificially inseminated to a bull on farm 1. One of the six aborted at three months but was negative to the blood test. The owner was of the opinion that this was due to insemination at very late heat, the heifer having been bred 48 hours after the heat period began. Because of an accident later, this animal was slaughtered.

There were five other commercial farms which requested artificial insemination for the same reason as farm 8. These farms are listed as Nos. 11, 13, 23, 26, and 28 in the table.

The use of artificial insemination in these cases could have been dispensed with and the cows inbred or not bred at all until a suitable sire was available, but the herd owners seem to feel that they have gained some advantage by resorting to artificial service by another bull. The value of this procedure to avoid inbreeding is another question which can best be answered by the breeders.

DISCUSSION

The cases reported here indicate that artificial insemination in private practice is of value in many instances. The results obtained are considered satisfactory where fertile subjects were offered for insemination. There were 148 pregnancies resulting from the insemination of 174 cows, and a total of 262 inseminations were made. A closer analysis of these figures shows how important it is to have fertile cows with which to work. One hundred and six cows conceived to the first service, 33 to the second, and nine to the third. The 148 cows which conceived required a total of 199 services. There were 63 services made on 26 cows which did not conceive. Eleven of these were rebred naturally and did conceive, but 15 were sterile. These wasted inseminations add to the grand total and increase the ratio of inseminations to pregnancies. The ratio was 1 pregnancy to 1.77 inseminations. This ratio, for some reason, is customarily accepted as a necessary figure to include in a summary on artificial insemination, but it is misleading because there are many factors which can influence the ratio. For example, on farm 5 the ratio was 1:1.3 and on farm 2 the ratio was 1:5. The average ratio of the two herds was 1:2.2, which does not apply to either herd and serves no useful purpose. An average ratio, however, can lead a farmer to believe that he can actually figure out how many services it will take to impregnate

his cows, and when it does not work out accurately, he is apt to be critical.

The writer has been making a practice of carefully examining and reviewing the breeding history of each subject for artificial insemination and if there is any reason to believe that the cow might be a difficult breeder, no attempt to inseminate her is made. In this way many cases are found where the cow is sterile and the owner was under the misapprehension that artificial breeding would correct the condition.

In conclusion, it may be said that artificial insemination meets a certain demand by commercial dairy farms as well as small farmers. As an aid in the control of trichomoniasis it has been found to be an effective means of preventing infection of a clean bull, and in cases where temporary breeding deficiency in a bull interrupts the breeding schedule, the schedule can be maintained until the deficiency is overcome.

Artificial insemination to an outside bull as a means of controlling Bang's disease in an infected herd was not proved to be satisfactory.

References

[1]Artificial insemination. Editorial in Jour. A.V.M.A., xcv (Dec. 1939), 753, pp. 733-734.

[2]Vet. Pract., xx (Dec. 1939), 12, p. 2.

[3]Williams, W. L.: Diseases of the Genital Organs of Animals. (W. L. Williams, Ithaca, N. Y., 1939.)

[4]Winters, L. M., *et al:* Artificial insemination of farm animals. Univ. of Minnesota Bul. 336.

$215,000,000 for Research

During 1939, industry spent about $215,000,000 for all types of research, and employed 32,000 scientists and engineers. In addition, 16,000 technical assistants and clerks were given employment.

One accomplishment that will save the railroads approximately $2,000,000 annually is the development of a new chemical that reduces the corrosive action of the brine that drips from refrigerator cars onto rails, bridges and other equipment. Another accomplishment of the year was the development of new industrial plastics from sawdust, alfalfa and wood pitch.—*Newsweek*, January 22, 1940.

Expiration Dates of Biological Products

The time beyond which a biological product is supposed to begin losing a part or all of its specified virtue is called the expiration date. The date for the various products is fixed by regulations. The dates are set for human medicine by the treasury department and for veterinary medicine by the U. S. Department of Agriculture, more specifically, by the U. S. Public Health Service and the U. S. bureau of animal industry, respectively.

An antitoxin with an excess potency of but 20 per cent is outdated in one year; of 30 per cent, two years; of 40 per cent, three years; and of 50 per cent, four years. Immune serums are given a potency tenure of six to 18 months and nonimmune serums, three years from the date of manufacture. The potency life of a toxoid is set at two years and most of the bacterins, at 18 months.

These are the figures fixed for human products. Veterinary products are subject to even stricter regulations in some instances. Hog-cholera virus, because of the importance of its disease-producing property, is given a tenure of but 90 days.

The time between the date of manufacture and the date of issue, unless otherwise provided, which shall not be more than three months, may, however, be prolonged by keeping products under stated degrees of refrigeration.

The variability of temperatures to which veterinary biological products are exposed necessitates close attention to expiration dates. The work of Ray and Whipple* on phenolated hog-cholera virus demonstrates the importance of this factor in the practice of veterinary medicine.

*Jour. A.V.M.A., xcv, September 1939, pp. 278-282.

Some Observations on the H-Ion Concentration of Cow's Milk During Estrum*

By ALBERT L. KLECKNER, Ph.D.
Philadelphia, Pa.

DURING the past several years a number of workers have reported upon the effect of various physiological conditions of the cow on the reaction of the milk to the more common milk tests used to diagnose catarrhal mastitis.

The stage of lactation and the age have been shown by Russell and Hoffmann,[1] Steck,[2] Seelemann,[3] Klein and Learmonth,[4] Hastings and Beach,[5] Klein, Kleckner and Scheidy[6] and others to influence the values of such milk tests as the leucocyte count and pH and chlorine, causing many of them to exceed the limits generally considered normal for milk.

The observations recorded here point to another physiological condition in the cow which appears to bear an influence on the pH of milk.

Quarter samples of milk were obtained from eleven purebred Guernsey cows at different stages of the same lactation period for a period of nine or ten months. Seven of the eleven cows were in the first or second lactation period, and the others were in the fifth lactation or later. Samples were obtained from each cow during at least two estrual periods, and in four cows there were four or more estrual periods covered in the same lactation. In all, 448 quarter samples were collected, 156 of which were obtained on the days that the animals showed evidence of estrum.

None of the cows showed any symptoms of disease. The foremilk was examined on a strip cup before each milking, and at no time during the period covered in this report was any abnormality, such as flakes, watery or off-colored milk, reported. Quarter samples of milk from each cow were plated out monthly on ox-blood agar. At no time during this period were mastitis streptococci or mastitis staphylococci recovered from the plates.

As soon as the milk samples were collected, they were taken to the laboratory for testing. The lapse of time between sampling and testing was generally less than 30 minutes.

The Brown[7] method, in which a La Motte hydrogen-ion outfit is employed, was used for determining the pH values of the milk samples. This method is a colorimetric comparison. One loopful (or two) of milk is mixed with seven or eight drops of neutral distilled water in small glass cells, one drop of brom-thymol-blue indicator added and the color compared with standard colors in glass cells of corresponding size.

Of the 156 quarter samples collected during estrum, 39 (25%) had a pH of 6.9 or 7, 61 (39%) had a pH of 6.8, and 56 (36%) fell in a pH range from 6.4 to 6.7, inclusive. On the other hand, twelve (4%) of the 292 quarter samples collected at a time when there were no symptoms of estrum had a pH of 6.9, 46 (16%) had a pH of 6.8, and 234 (80%) had a pH of 6.7 or less.

When the milk samples were collected on the day that estrum was most evident, all eleven cows had one or more quarters in which the pH of the milk was 6.9 or 7; all had one or more quarters in which the pH of the milk was 6.8, and ten (91%) had one or more quarters in which the pH of the milk was 6.7 or less. On the other hand, when milk samples were obtained from the same eleven cows at a time when they showed no signs of estrum, only five (45%) had one or more quarters in which the pH of the milk was 6.9, ten (91%) had one or

*From the School of Veterinary Medicine, University of Pennsylvania. This investigation was supported partly by the federal bureau of animal industry and partly by the University of Pennsylvania, the latter being assisted by contributions from herd owners and milk distributors.

more quarters in which the pH of the milk was 6.8, and all of the cows had one or more quarters in which the pH of the milk was 6.7 or less.

In a few instances consecutive daily samples of milk were obtained, starting three to four days before the animals were expected to show symptoms of estrum and continuing for several days afterward. This was done to determine whether the effect on the pH, if due to estrum, was gradual or whether the pH was increased only on the day the symptoms were most evident. The results indicated that the pH was increased only at the height of estrum and dropped back to a value similar to that found previously by the next day.

There was a reduction in the daily milk production in all of the cows at the height of estrum during one or more of the estrual periods covered. In seven (64%) of the cows this reduction in daily production occurred at the height of estrum during each estrual period. The remaining four cows (36%) showed this reduction on the day estrum was most evident in ten of the 15 estrual periods covered. The drop in production ranged from 0.2 to 12.8 pounds* in the former cows and from 0.2 to 3 pounds in the latter group. The mean reduction was 2.5 pounds and 1 pound, respectively.

In the eleven cows there were 34 estrual periods in which the production had decreased on the day estrum was most evident. Of the 136 quarter samples obtained during these periods, 33 (24%) had a pH of 6.9 or over. There were five periods during which no reduction in production occurred. Of the 20 milk samples obtained at this time, six (30%) had a pH of 6.9 or over. There was no correlation between the extent of reduction in the daily quantity of milk on the day that estrum was at its height and the pH of the quarter samples obtained at that time. The pH of the quarter samples obtained on the days when the cows showed the greatest decrease in the quantity of milk produced was not significantly higher or lower than the values obtained from the quarters on days when the production was only slightly affected.

The comparatively small number of cows observed, as well as the fact that the animals were all in the same herd and subjected to the same management, does not warrant the formation of any conclusions on the results obtained. Rather, this report is presented as an interesting observation and it is hoped that it may lead to further and more comprehensive investigations on estrum and its relation to the reaction of milk.

*Reduction was determined by averaging the total daily production for three days preceding the day estrum was most evident and subtracting the total production of the day that estrum was at its height from this figure.

SUMMARY

Quarter samples of milk were obtained from eleven purebred Guernsey cows over a period of nine to ten months and tested for pH. These cows were all free from symptoms of mastitis or other diseases, as determined by physical examination. No mastitis streptococci or staphylococci were found on the ox-blood agar plates from any of the samples during this period.

In all, 448 quarter samples were obtained, of which 156 were collected on the day that symptoms of estrum were most evident. Samples were obtained from each cow during at least two estrual periods in the same lactation, and in four cows there were four or more estrual periods covered in the same lactation. Seven of the eleven cows were in the first lactation period and the others were in the fifth lactation period or later.

Twenty-five per cent of the quarter samples collected at the height of estrum had a pH of 6.9 or over, 39 per cent had a pH of 6.8, and 36 per cent had a pH of 6.7 or less.

Only 4 per cent of the quarter samples which were collected in the intervals between estrual periods had a pH of 6.9 or over, 16 per cent had a pH of 6.8, and 80 per cent had a pH of 6.7 or less.

In respect to cows on the day estrum was most evident, all of them had one or more quarter samples with a pH of 6.9 or over, all had one or more quarter samples with a pH of 6.8, and ten (91%) of the cows

had one or more quarter samples with a pH of 6.7 or less.

In the samples collected in the intervals between estrual periods, only five (45%) of the cows had one or more quarter samples with a pH of 6.9 or over, ten (1%) had one or more quarter samples in which the pH was 6.8, and all of the cows had one or more quarter samples in which the pH was 6.7 or less.

The pH apparently did not rise gradually over a period of several days, but showed this rise only at the height of estrum, and dropped back to normal by the following day.

In the majority of estrual periods (34 out of 39) observed in the eleven cows, there occurred a reduction in total production on the day estrum was most evident, ranging from 0.2 to 12.8 pounds. There was apparently no relation between this reduction in milk flow and the increase in pH. The quarter samples collected on the day when estrum was at its height showed similar percentages of samples with a pH of 6.9 or over, or 6.8 or 6.7 or less, regardless of whether or not there was a reduction in total production on that day.

These results, although too few in number to be of conclusive significance, seem to point out another physiological condition of the cow that affects at least one of the more common milk tests used to detect mastitis.

References

[1]Russell, H. R., and Hoffmann, C.: Distribution of cell elements in milk and their relation to sanitary standards. Univ. of Wisconsin Agr. Exp. Sta. Ann. Rpt. (1907), p. 231.

[2]Steck, W.: Die Latente Infektion der Milchdruse vorn path.-physiol. und milchwirtsch. (Standpunkt, M. and H. Schaper, Hanover, 1930.)

[3]Seelemann, M.: Die Streptokokken-infektionen des Enters. (M. and H. Schaper, Hanover, 1932.)

[4]Klein, L. A., and Learmonth, R.: Significance of streptococci, and high leucocyte count, H-ion concentration and chlorine content in cow's milk. Jour. A.V.M.A., lxxxvi (1935), n. s. 39 (5), p. 600.

[5]Hastings, E. G., and Beach, B. A.: The production of milk of abnormal composition by animals free from udder streptococci. Jour. Agr. Res., liv (1937), p. 199.

[6]Klein, L. A., Kleckner, A. L., and Scheidy, S. F.: Comparison of the results of certain milk tests with the condition found on physical examination of the cow. Univ. of Pennsylvania Bul., Vet. Ext. Quart., xxxix (1938), p. 3.

[7]Brown, J. H.: The colorimetric determination of the hydrogen ion concentration of small amounts of fluid. Jour. Lab. & Clin. Med., ix (1924), p. 239.

Helium

Once a rare element costing $2,000 per cubic foot, helium is now obtainable at one cent per cubic foot. The United States is the main source of supply. Being lighter-than-air gas and noncombustible, it is a precious element in aeronautics. Recently, helium has come into use in the practice of medicine with a promising future in an important rôle. Mixed with 20 per cent of oxygen, this gas has been found to give immediate relief to victims of asthma.

Hypertonic Salt Solution in Azoturia

Among the medicinal treatments which appear to have a certain amount of value for azoturia is the intravenous injection of sodium chloride as early as possible after the onset of the attack. The dose recommended is 1,500 cc. (3 pints) of a 10 per cent aqueous solution. The effect is the lowering of glycemia and azotemia and augmentation of the red cell count. Other effects attributed to this treatment are decreasing albuminuria and coloration of the urine, and stepping up of renal secretion. Zieba (*Rev. de Méd. Vét,* October 1938) pronounced this the treatment of choice. The treatment is of no value in the advanced phase.

Lou Gehrig's Ailment Treated with Vitamin B_1

The muscle-wasting affliction which cut short the brilliant career of Lou Gehrig, baseball's "iron man," is said to be associated with vitamin B_1 deficiency and, says *Newsweek,* "Dr. W. J. McCormick revealed that Gehrig is taking tablets of that substance. . . This vitamin is known as an aid in the treatment of nerve-affecting ailments." The report goes on to say that a Canadian veteran of the World War suffering from a similar trouble showed improvement after taking vitamin B_1 pills for ten weeks.

Vitamin E deficiency in chickens causes degeneration of the circulatory system of the chick embryo.—*Adamstone* (1931).

Diseases of Feeder Cattle*

By W. S. O'NEAL, D.V.M.
St. Charles, Mo.

THE OWNERS of feeder cattle should be encouraged to build small pens, crowding gates, and stocks to facilitate the handling of the herd. Such investments pay dividends in time saving and promote safety to both men and animals. The veterinarian should be amply equipped for the proper restraint of individuals.

HEMORRHAGIC SEPTICEMIA

So-called hemorrhagic septicemia or shipping fever of feeder cattle has been discussed so ably by Dr. Aitken (see page 300) that no further mention of this condition will be made here. It may be stated, however, that sulfanilamide has been of distinct value in treating this disease.

BLACKLEG

Blackleg occurs but rarely in St. Charles and St. Louis counties of Missouri. Five sporadic cases have been seen by the writer in 13 years of practice. Great care has been exercised in the disposal of the carcass of each case in an endeavor to continue as long as possible our comparative freedom from this disease. In each case all bovine animals on the exposed premises were treated with blackleg bacterin.

INFECTIOUS KERATITIS

Observations of keratitis in feeder cattle lead to the belief that there are infectious and noninfectious types of this disease. However, in the handling of this condition no attempt is made to differentiate between the two types. All are treated with keratitis bacterin; the affected animals are separated from the nonaffected, and two or three drops of 5 per cent mercurochrome or 5 per cent argyrol are placed on the diseased eye once daily.

ACTINOMYCOSIS

Surgical procedure is rarely employed in actinomycosis. Potassium iodide is seldom used. If the tumor is circumscribed and free of vital structures, the area is shaved and 15 to 25 cc. (0.5 to 0.8 oz.) of formalin injected into the abscess. The amount of formalin to be injected is judged by the pressure on the syringe and within the tumor. Full pressure should be obtained. The formalin embalms the tumor and, gradually, a separation occurs between the embalmed tumor and the surrounding healthy flesh. The line of separation should be treated with an antiseptic oil. In about three to four weeks the tumor may be removed by torsion, and the resulting open wound treated as such.

ANAPLASMOSIS

Animals affected with anaplasmosis should be segregated. Liberal use of fly spray on the affected animals is recommended on the ground that further spread of the disease is thus partially limited. Sodium cacodylate is used, 12 Gm. (180 gr.) being the initial dose for a 409-kg. (900-lb.) animal. This is followed with a commercial powder containing arsenic and willow bark. The grain ration should be liberal to promote complete recovery.

COCCIDIOSIS

Coccidiosis sometimes becomes a grave problem in feeder cattle. A 50 per cent infection has been observed following the feeding of alfalfa hay from a new source. Affected animals are segregated and their feces hauled to some part of the farm not used for the production of forage for cattle.

The affected animals are given salicylic and tannic acid, equal parts, 15 to 120 cc. (0.5 to 4 oz.), according to the size of the animal and the severity of the condition. The acids are suspended in a quart of water and given as a drench. Three doses daily are given until the condition abates. It is beneficial to follow this treatment with sun-cholera cure, prepared according to the fol-

*Presented before the Section on General Practice at the 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1939.

lowing formula, until the feces are about normal:

Sun-Cholera Cure

Spirits of peppermint
Spirits of camphor
Tincture of rhubarb
Tincture of capsicum
Tincture of opium aa
M.
Sig. 1 dr. to 1 oz., acording to the size of the animal, in water, two to three times daily.

DIGESTIVE PROBLEMS

Impaction of Rumen.—From one to three pounds of magnesium sulfate in three to five gallons of water and an antiferment are administered with the stomach tube. Rumenotomy has fallen into disuse. In cases where a rumenotomy is indicated, the following procedure is recommended. A large cannula is introduced into the rumen. A double-looped baling wire is inserted through the cannula and used as a pump to break up the impacted mass and to remove much of the contents of the rumen. Water is pumped in through the cannula to aid in the breaking up and pumping out of large quantities of the impacted mass. After the mass is broken up, the above-mentioned purgative is pumped into the rumen while the cannula is still in place.

Bloat.—It is seldom necessary to introduce a cannula into the rumen for the relief of so-called dry bloat. This condition is usually relieved by the use of the stomach tube. For the relief of so-called wet bloat the procedure is the same as outlined for impaction of the rumen except that it is seldom necessary to pump any water into the rumen.

Traumatic Gastritis.—No treatment is attempted in feeder cattle. Early diagnosis is important so that the animal may be sold for slaughter while in a marketable condition.

Enteritis.—The treatment for this condition is practically the same as that outlined for coccidiosis.

MISCELLANEOUS PROBLEMS

Foot Rot.—Foot rot is a serious problem in feeder cattle. The amount of handling necessary in the treatment of this condition often results in neglect. In herds showing a high incidence of this condition, a concrete tank with 4-in. walls should be built around the water tank. Ten to 12 pounds of copper sulfate is dissolved in 40 gallons of water and the solution placed in the concrete tank. Thus, all of the animals bathe their feet in this solution and a material reduction in the spread of the disease is soon evident.

Ocular Tumors.—Tumors of the eyeball are seldom relieved surgically; the owner prefers to market the animal. Extirpation of part or all of the membrana nictitans is practiced for neoplasms upon that structure.

Ringworm.—Ringworm usually clears up after a few applications of tincture of iodine. Equal parts of tincture of iodine, glycerin and chloral hydrate seem to be more efficient than tincture of iodine alone.

Sweet Clover Hemophilia.—This condition is of prime importance during the dehorning and castrating seasons. Never fail to inquire as to the recent feeding of sweet clover hay. Never attempt any routine operation on cattle recently fed on sweet clover; it is better to wait until they have been on other feed for two or three months than to find yourself beset with a large number of bleeding steers.

Nutritional and Mineral Deficiencies.—Observation leads one to believe that in this category we are but entering upon a field of usefulness to the livestock industry. The subject is so large and the ramifications of these deficiencies so varied that no discussion will be attempted within the limited scope of this paper. It is believed that nutritional and mineral balances are an integral part of all pathological conditions and should receive our unending attention.

It takes three weeks of her annual salary to pay the taxes on the cosmetics of the average working girl, says *Chemical Industries.*

About 30 million animals are slaughtered annually in the United States without any supervision as to the wholesomeness of their meat. Of these, it was estimated by Koen (J. S.) that 160,000 should have been condemned as unfit for human consumption.

Lead-Arsenate Poisoning of Sheep and Cattle*

By ERNEST C. McCULLOCH, D.V.M., M.A., Ph.D., and J. L. ST. JOHN, Ph.D.
Pullman, Wash.

APPROXIMATELY 44,000,000 pounds of lead arsenate are used in agriculture each year.[1] With such quantities being applied in rural areas, and not infrequently by laborers who do not fully appreciate its poisonous properties, it is inevitable that substantial amounts will contaminate the forage of domestic animals. Yet, the reported deaths directly traceable to lead-arsenate poisoning are relatively few and sporadic,[2] even in such areas as the Yakima Valley, where more than 3,500,000 pounds of lead arsenate are applied as orchard sprays annually.[3] This is largely because livestock men and orchardists have learned from past experiences the danger of orchard forage for live stock. Sometimes, however, sheep and cattle are allowed to feed upon forage that has been heavily contaminated with lead-arsenate sprays. Occasionally, the threshold of toxicity is exceeded and losses follow. Usually, such losses occur when exceedingly hungry animals consume large quantities of heavily sprayed materials.

POISONING OF 1,000 SHEEP NEAR ORONDO, WASH.

On October 14 and 15, 1936, a flock of somewhat more than 1,000 sheep were driven from the summer range through rather barren country and maintained on a sand bar near Orondo, Wash., the night of the 15th. The next day they were ferried across the Columbia River in groups of approximately 200 head and allowed to fill upon the lush forage under the apple trees. On the morning of the 17th, the herder could not get the sheep to leave the bedground, and the first death occurred on the evening of the same day. On the following morning five were dead; by noon 15 and by late evening 60 had succumbed. On the morning of the 19th, 200 were dead, and by evening the number had increased to 500. The following day approximately 200 died, and 50 the next day. On October 25, the 319 survivors were removed by truck to Ellensburg, Wash. Losses were still occurring on October 31, and by January 1, 1937, but few were left.*

Before the arrival of one of the writers on October 23, a preliminary diagnosis of anthrax was made and most of the sheep already dead were burned.

Autopsies were made on sheep immediately after death, and upon moribund survivors. All showed evidence of a profuse diarrhea. Erosions were found in the lining of the rumen and reticulum, and the intestinal walls were highly inflamed. The mesenteric lymph glands were congested and swollen and in some sheep peritonitis had developed. More than one half of the sheep examined showed some degree of pneumonia, a condition later found to occur in sheep which had been experimentally poisoned with lead arsenate.

A diagnosis of acute lead-arsenate poisoning was made and it was suggested that the pelts be salvaged from the dead sheep which had not been incinerated. Several samples were taken from different parts of the rumen and reticulum of one of the last sheep to die. These were analyzed for lead and arsenic and found to contain over 0.05 per cent lead and more than 0.06 per cent arsenic calculated as As_2O_3. Assuming a rumen capacity of 15 liters (4 gals.),[4] which is rather small in view of the fact that these animals had good fills, this would indicate the presence of at least 30 Gm. (1 oz.) of lead arsenate. Since four days had elapsed and the sheep had absorbed sufficient poison to cause death and probably had eliminated some of the lead arsenate, it is conceivable that a considerably greater amount was consumed. How much greater

*Published as scientific paper No. 415, College of Agriculture and experiment station, State College of Washington; contribution from the divisions of veterinary science and chemistry.

*This report was obtained from the owner and given to the writers by Peter MacKintosh of Yakima, Wash.

quantities were consumed by the sheep which died one and two days earlier is not known, nor is it known how much less was consumed by those sheep that did not die. It is reasonable to assume, however, that the last to die probably consumed only slightly more than the minimum lethal doses. Bacteriological cultures and direct blood smears of these sheep proved negative.

The orchard in which the sheep had grazed was examined closely. The forage under the trees was chalky in appearance because of the lead-arsenate spray that had adhered to it. Representative samples were collected for analysis, which revealed the presence of 0.58 per cent arsenic (As_2O_3) and 1.44 per cent lead. This would amount to 2.63 Gm. (40.6 gr.) of arsenic and 6.55 Gm. (100.8 gr.) of lead per 480 Gm. (1 lb.). In passing, it is of interest to note that these amounts are approximately 4,060 and 4,030 times the present Food and Drug Administration tolerances for arsenic and lead, which are 0.01 and 0.025 grain, respectively, per pound of food.[5]

The amount of lead arsenate applied as a spray to this particular orchard was high, but not abnormally so for apple trees in this region. About 273 kg. (600 lbs.) of lead arsenate per acre was applied in nine sprays between May 10 and August 13. Between the latter date and October 20, the precipitation at Wenatchee, 17 miles away, was 0.53 inches, and approximately the same at Orondo.

In previous years it was the custom to permit sheep to graze the forage in orchards in return for their ability to clean out weeds. Only occasionally had losses occurred, and usually these were attributed to infectious diseases rather than to lead-arsenate poisoning. These orchards, however, were grazed later in the fall, and usually after more rain had fallen. In addition, the sheep were not extremely hungry when turned into the orchards. The increased amount of lead arsenate now being applied, together with the superior adhesives[6] now employed, probably will greatly increase the hazard of grazing these orchards at any time during the fall or winter.

The diagnosis of lead-arsenate poisoning in these animals also was made by Dr. MacKintosh. It is interesting to note that this particular region long has been considered an "anthrax center" and there is a lengthy history of sudden deaths of sheep which have been ferried across the river at this point. Dr. MacKintosh reports that during the twenty years he has been familiar with this region, no case of anthrax has occurred. It appears probable that lead-arsenate poisoning of very hungry sheep that have been allowed to engorge upon sprayed forage has been responsible for these losses.

LOSS OF 23 FEEDER STEERS AT ZILLAH

On October 4, 1937, approximately 80 steers, which had been shipped from Montana, were unloaded at Zillah, Wash., and were immediately turned into a pear and apple orchard. Within two days 17 were dead and most of the remainder had a profuse, very dark, and in some cases a bloody, diarrhea and seemed dejected in appearance. Although somewhat similar symptoms follow engorgement upon any fruit, subsequent findings indicate lead-arsenate poisoning to have been an important factor in these deaths.

Autopsies on the last to die and on a moribund steer on October 7 revealed areas of erosion in the lining of the rumen and an intense enteritis. A considerable amount of pear leaves and twigs was found among the rumen contents. Samples of the mixed rumen contents were taken for analysis. They contained 0.008 per cent arsenic (calculated as As_2O_3). Assuming a stomach content of 136 kg. (300 lbs.),[7] which would appear to be reasonable in an engorged steer, this would indicate the presence of over 38 Gm. (1.25 oz.) of lead arsenate. Two family cows had been maintained in the orchard since late summer and showed no untoward effect.

A careful investigation was made of the orchard in which the steers had been placed. Areas of alfalfa in the centers between the rows apparently had supplied the food for the family cows. The steers, however, were noted by the attendant to be very hungry upon arrival. The relatively small amount

of alfalfa apparently failed to attract the attention of some, which were observed to feed upon the leaves of the lower branches. Also, there were indications that these animals had licked the trunks of the apple and pear trees. Analyses of the leaves from the pear trees from one orchard revealed about 2 Gm. (31 gr.) of arsenic (As_2O_3) per 480 Gm. (1 lb.) of air-dry foliage, which is equivalent to about 7 Gm. (108 gr.) of lead arsenate per 480 Gm. Foliage from another orchard carried about half this much spray. Analysis of the bark also demonstrated the presence of arsenic.

While only two more animals died within the next week, several failed to make normal gains with subsequent feeding and the mortality during the winter was higher than usual.

In explaining the good health enjoyed by the family cows permanently pastured in the orchard, it is not necessary to assume an acquired increased resistance to lead arsenate, since they apparently fed almost entirely upon the narrow strip of alfalfa in the center of the rows, which had been subject to much less spray than the trees. The steers, however, engorged upon the leaves of the heavily sprayed pear trees.

ADDITIONAL CASES

According to MacKintosh of Yakima,[8] many acute cases have resulted from the animals' gaining access to containers of lead arsenate, which is very palatable to them, or from their being allowed to graze in orchards which have been sprayed very recently. With the education of the orchardists to the hazards of exposures of this magnitude, the losses have decreased.

Horses appear to be more susceptible to chronic lead poisoning than are cattle. The condition known as "orchard-horse disease" has been described by Kalkus.[9] Horses not otherwise exposed to spray sometimes are poisoned from hay.[10] Hay from old orchards, which have received large amounts of spray, may poison after six to eight months of feeding, while hay from young orchards may be fed for as long as six years before producing symptoms.

EXPERIMENTAL FEEDING OF LEAD ARSENATE TO SHEEP*

In order to establish more definitely the toxicity of known quantities of lead arsenate for sheep, healthy young sheep weighing between 36 and 45 kg. (80 and 100 lbs.) were given weighed amounts of lead arsenate in gelatin capsules. The daily dose was divided into two equal portions and given morning and night.

The first series of three sheep were given 2 Gm. (31 gr.) of lead arsenate daily. One was maintained on a basic ration consisting of 678 Gm. (22.5 oz.) of hay, 204 Gm. (7 oz.) of rolled oats and 150 Gm. (5 oz.) of rolled barley with salt *ad libitum*. The other two received about 10 per cent less of this basic ration with 960 Gm. (2 lbs.) of apples per day to provide succulence.

Kidneys and section through ribs of a sheep fed 17.5 Gm. (4.5 dr.) of lead arsenate over a period of 35 days. Note the extremely hard kidneys and bone marrow.

The sheep receiving only the basic ration and one of the sheep receiving apples in addition died after 3½ days, after having received 7 Gm. (108 gr.) of lead arsenate. They refused almost all food after the second day. Autopsy revealed hemorrhagic erosions of the rumen wall, and these extended into the reticulum. A severe enteritis was present, especially in the region of the duodenum. The lungs were pneumonic. The remaining sheep survived for seven days and received 15 Gm. (0.5 oz.) of lead arsenate. Postmortem lesions were essentially similar to those of the other

*The feeding experiments, methods of chemical analysis and results are being reported in greater detail elsewhere.

two sheep, except for a metallic sheen to the kidneys.

Following the administration of the first dose of lead arsenate to these sheep, the intake of feed and water was sharply reduced. Arsenic was eliminated in the urine, although the feces contained a lesser amount. Upon autopsy from 44 to 48 per cent of the total amount of arsenic fed was accounted for by analyses of the contents of the digestive tract and from 12 to 14 per cent in the liver and kidneys.

More lead was eliminated in the feces than in the urine. Upon chemical analyses of the digestive tract, from 60 to 77 per cent of the total lead administered was accounted for. The liver and the kidneys also contained small amounts.

Since the amounts administered in this experiment killed too quickly to yield the desired information, another series was started in which three pairs of sheep were fed 1, 0.5 and .25 Gm. (15, 8 and 4 gr.) of lead arsenate per day. One of each pair received the basic ration while one received apples in addition. A seventh sheep was included as a control.

The sheep receiving 1 Gm. of lead arsenate per day and apples in the ration died in six days, and the other sheep on this level but receiving only the basic ration died in seven days. The findings at autopsy were similar to those previously described.

One sheep on the basic ration and receiving 0.5 Gm. of lead arsenate per day died after 35 days. As with the other sheep which survived long enough for the phenomenon to be observed, the erythrocyte count was found to rise and then to fall to low levels a few days before death, in this case from approximately 8,000,000 before the feeding of the lead arsenate to 13,000,000 on the tenth day to 4,200,000 on the 27th day. The leucocyte count fluctuated within normal limits until bacterial infection set in, when it rose sharply in most cases. A lead line was observed on the 14th day. The appetite diminished on the 18th day, and four days later the sheep refused almost all food, continuing to do so until death. On the 26th day a foul diarrhea appeared and hemoglobinuria was observed the day before death. After 35 days the animal died in a cachectic state.

On post mortem a distinct lead line was observed. There were no erosions on the mouth, esophagus, or in other portions of the digestive tract. Only a moderate degree of enteritis was present. The heart showed evidence of fatty degeneration, the lungs were congested and pneumonic. The spleen was ruptured and swollen with the pulp distinctly darkened. The kidneys were blue-black, having a metallic sheen. They were so soft that removal without breaking was difficult. The marrow of the flat bones was black. Upon exposure to the air the marrow showed a tendency to lighten to dark red.

One of the sheep receiving .25 Gm. of lead arsenate per day died at the end of 35 days with symptoms and postmortem lesions very similar to those of the others receiving lethal amounts. The other sheep survived .25 Gm. of lead arsenate per day for 94 days, when the feeding was discontinued. When slaughtered ten months later, this sheep was in excellent condition and showed no evidence of having consumed 23.5 Gm. (0.8 oz.) of lead arsenate. Of especial interest was the fact that the bones showed no evidence of darkened marrow.

Is Chronic Arsenic Poisoning Responsible for Black Cutters?

The black marrow of the flat bones of some of the sheep fed lead arsenate for considerable periods may throw additional light on the condition known as black cutters. Bull, Olson and Kammlade[11] previously have done work on this phase of the problem.

Review of the Literature

The amounts of arsenic and lead that some of the exposed animals survived was of a magnitude so different from the official tolerances for these substances for humans that other data were sought.

Seddon and Ramsay[12] fed 36- to 45-kg. (80- to 100-lb.) sheep various amounts of different arsenic and lead compounds. Their experimental animals fed single doses of 2.6 and 7.8 Gm. (40 and 120 gr.) of lead

arsenate survived while one fed 3.9 Gm. (60 gr.) succumbed on the twelfth day and one fed 11.7 Gm. (180 gr.) was killed in a moribund condition on the sixth day. Sheep fed 6.6 gr. of arsenous acid lived, while of two fed 13.3 gr., one died on the fifth day and the other survived. Doses of 1.3, 1.95 and 3.9 Gm. (20, 30 and 60 gr.) killed on the second or third day.

The lead compounds were less toxic, 11.7 Gm. (180 gr.) of lead oxide killing on the eighth day while 4 Gm. (60 gr.) of lead sulfate failed to kill. Sheep were killed by 17.5 Gm. (270 gr.) of lead acetate but survived 14 Gm. (216 gr.). For sheep they estimate the following as fatal doses:

	Grams	Grains
Sodium arsenite	0.908	14
Paris green	1.1	17
Arsenous acid	1.04	16
Lead oxide	11.7	180
Lead acetate	18	275

Husband and Duguid[13] reported that the grazing of cattle on grass to which sodium arsenate had been applied at the rate of 720 Gm. (1.5 lbs.) per acre in combating grasshoppers resulted in their death in 96 hours from arsenical poisoning.

Thomas and Shealy[14] found that chickens might consume as much as 13 gr. of lead arsenate daily for 60 days without suffering ill effects.

Wilson and Holmes[15] were unable to get chickens to eat sufficient arsenic grasshopper bait to prove toxic. The formula they used was:

Sawdust	100 Gm.
Whey	30 Gm.
Arsenic trioxide	4 Gm.

When bran was substituted for the sawdust, two out of 24 chickens on experiment ate enough to die from the feed. One consumed an estimated 3 Gm. (46 gr.) of arsenic and the other approximately 1 Gm. (15.4 gr.). The others ate more sparingly and gradually went off feed, although a number ate food containing 2 Gm. (31 gr.) of arsenic over a period of three days and survived.

There is a general belief that a tolerance to arsenic is readily acquired and that the long-continued consumption of moderate amounts of arsenic is not deleterious. This view does not appear to be justified by clinical and experimental evidence. Talber and Tayloe,[16] working with white rats, found that arsenic or lead, or both, when fed in quantities larger than the equivalent to 0.04 gr. for about 175 days, seemed to have an injurious effect on the offspring by decreasing the weight and the ability of the females to produce and breed young. These same investigators found that the spray insecticides do not have an acute toxic effect upon albino rats even when used in amounts of 200 times the Food and Drug Administration tolerance of 0.1 gr. of arsenic and 0.025 gr. of lead per pound.

It is interesting to note that many valued foods contain much more arsenic than the 0.01 gr. per pound tolerance for foods. White[17] reported the arsenic (As_2O_5) content of fresh codfish as from 0.014 to 0.038, shrimp from 0.017 to 0.077 and lobster as high as 0.126 gr. per pound.

RUMINANTS ARE RELATIVELY RESISTANT TO LEAD POISONING

Perhaps the lower incidence of plumbism in animals consuming lead-contaminated forage than in people exposed to lead in the industries may be explained by the difference in the ways in which exposure occurs. Lanza[18] stated:

> Most of the industrial exposure arises from dust and fumes that are breathed into the lungs and upper respiratory tract, where absorption and excretion involve entry into the systematic circulation. Lead that is ingested may be excreted unchanged and, even if absorbed, may be carried to the liver and excreted in the bile.

Cardiff[19] believes that the danger to horses from eating hay from sprayed orchards comes from the inhaled spray residues rather than from the lead and arsenic consumed.

Except in the case of animals receiving large amounts of lead in contaminated water or acute poisonings from eating paint, ruminants appear to be rather tolerant to lead.

ARE LEAD-ARSENATE SPRAYS LESS TOXIC THAN LEAD ARSENATE?

From the foregoing data it is apparent that the amounts of lead arsenate consumed by animals on sprayed forage and estimated

from chemical analyses of the stomach contents are substantially greater than the amounts that sheep survived when fed capsules containing weighed amounts of lead arsenate. The differences in amounts tolerated become greater when the fact that slightly less than one half of the arsenic and less than two thirds of the lead administered to an animal could be accounted for by analyses of the stomach contents.

The possibility is suggested that lead-arsenate sprays on living foliage undergo some change which renders them less soluble in the digestive tract and therefore less toxic. As has been shown by O'Kane, Hadley and Osgood,[20] arsenic ingested in the form of lead arsenate is less toxic than when ingested as white arsenic. Also, there is evidence that arsenic, in the form of sea food, may be almost entirely eliminated from the body with practically no absorption into the system.

Coulson, Remington and Lynch[21] found that rats fed shrimp containing 17.7 parts of arsenic per million stored only 0.13 mg. of arsenic in three months while in a parallel experiment rats which had a similar amount of As_2O_3 added to their diet stored 3.73 mg., or 28.7 times as much.

In view of the quantities of spray residue required to kill sheep and cattle, the observation of Mellor[22] that a dangerous dose of arsenic for a horse is 1.9 Gm. (29 gr.) and for a cow 0.64 Gm. (11 gr.) is of interest.

SUMMARY AND DISCUSSION

In most of the trials sheep died when experimentally fed sufficient lead arsenate that they obtained arsenic equivalent to approximately 2 Gm. (31 gr.), expressed as As_2O_3. Analyses of the rumen contents of animals poisoned from eating lead-arsenate-sprayed forage, however, indicated that much greater amounts of arsenic had been consumed. The possibility that the lead and arsenic were converted to some less toxic form is suggested. The lowered toxicity may be the result of decreased solubility within the digestive tract.

The darkening of the bone marrow observed in some of the lead-arsenate-fed sheep supports the suspicion that the feeding of arsenic-containing compounds may be responsible for the condition known as "black cutters."

References

[1]Stine, O. C.: Personal communication (1939).
[2]Reif, E. C.: Personal communication (1939).
[3]*Idem.*
[4]Sisson, S., and Grossman, J.: The Anatomy of the Domestic Animals (W. B. Saunders Co., Philadelphia, 1938), p. 479.
[5]U. S. Dept. Agr. Press Releases (Jan. 26, 1937; Sept. 19, 1938).
[6]Marshall, J., and Groves, K.: Further experiments with inverted or "dynamite" spray mixtures. Proc. Wash. State Hort. Assn., xxxiii (1937), p. 119.
[7]Sisson, S., and Grossman, J.: The Anatomy of the Domestic Animals (W. B. Saunders Co., Philadelphia, 1938), p. 456.
[8]MacKintosh, P. G.: Personal communication (1939).
[9]Kalkus, J. W.: Orchard horse disease. Amer. Jour. Vet. Med., xv (1920), p. 139.
[10]MacKintosh, P. G.: Clinical manifestations and surgical treatment of lead poisoning in the horse. Jour. A.V.M.A., lxxiv (1929), n. s. 27 (2), p. 193.
[11]Bull, S., Olson, F. C., and Kammlade, W. G.: Fowler's solution may be the cause of black cutters. Illinois Agr. Exp. Sta., 44th Ann. Rpt.
[12]Seddon, J. R., and Ramsay, A. A.: Toxicity of certain arsenic and lead compounds for sheep. N. S. Wales Dept. Agr., Vet. Res. Rpt. No. 6, pt. 3 (1933), p. 113.
[13]Husband, A. D., and Dugued, J. R.: The toxicity to grazing animals of grass sprayed with a solution of sodium arsenate. Rhodesia Agr. Jour., xxxi (1934), p. 25.
[14]Thomas, E. F., and Shealy, A. L.: Lead arsenate poisoning in chickens. Jour. Agr. Res., xlv (1932), p. 317.
[15]Wilson, J. F., and Holmes, C. E.: Effect on chickens of arsenic in grasshopper bait. Jour. Econ. Ent., xxix (1936), pp. 1008-1014.
[16]Talbert, T. J., and Tayloe, W. L.: Some results from feeding spray chemicals to albino rats. Missouri State Res. Bul. 183 (1933).
[17]White, W. B.: Poisonous spray residues on vegetables. Ind. Eng. Chem., xxv (1933), p. 621.
[18]Lanza, A. J.: Epidemiology of lead poisoning. Jour. Amer. Med. Assn., civ (1935), p. 85.
[19]Cardiff, I. D.: Observations with reference to arsenic on apples and other foodstuffs. Proc. Wash. State Hort. Assn., xxxiii (1937), p. 153.
[20]O'Kane, W. C., Hadley, C. H., and Osgood, W. A.: Arsenical residues after spraying. New Hampshire Agr. Exp. Sta. Bul. 183 (1917).
[21]Coulson, J. E., Remington, R. E., and Lynch, K. M.: Toxicity of naturally occurring arsenic in foods. Sci., lxxx (1934), p. 230.
[22]Mellor, J. W.: A Comprehensive Treatise on Inorganic and Theoretical Chemistry, Vol. 9 (1929), p. 43.

"Medical journals have been obliged by the war to reduce the number of pages," says the London correspondent to *The Journal of the American Medical Association.* Contributors have been requested to be "sparing of their words."

The Relation of Vitamins to the Nutrition of Farm Animals*

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TODAY there are at least ten dietary factors designated as vitamins which have been either partially or completely characterized chemically. These may be listed as follows: 1) Vitamin A, 2) vitamin B_1 (thiamin), 3) vitamin B_2 (riboflavin), 4) vitamin B_6 (rat antiacrodynia factor), 5) antipellagra vitamin (nicotinic acid), 6) chick antidermatitis vitamin (pantothenic acid), 7) vitamin C, 8) vitamin D, 9) vitamin E and 10) vitamin K.

The original vitamin B later proved to be a mixture of several dietary essentials which are now referred to collectively as the vitamin B group or complex. Those numbered 2 to 6 inclusive are all members of the vitamin B group. In addition to the above there are about a dozen other factors which have been suggested by the work of various investigators. Several of these are found in yeast and, as such, are also classed as part of vitamin B. Probably the most widely accepted of these additional members of the B group is factor W of Frost and Elvehjem.[1] Of those not belonging to the B complex the factor which prevents gizzard erosion in fowls is the best known and, according to Almquist and Mecchi,[2] is cholic acid or derivative. Just how many of these tentative dietary essentials will prove to be separate entities remains to be seen. It is possible that some of them are identical with those already recognized. Until more is learned concerning them, little can be said of their relation to the nutrition of domestic animals.

ALL HIGHER ANIMALS APPEAR TO REQUIRE VITAMINS A AND B_1

Some of the vitamins which have been definitely proved to exist are essential to farm animals, some apparently are not and still others have not been thoroughly studied in this respect. As far as is known, all of the higher animals require vitamin A. The same is thought to be true of vitamin B_1, or thiamin. However, in ruminants a considerable amount of this factor is formed in the gastrointestinal tract under the action of microörganisms. The same is probably true for other constituents of the B complex. Recently, McElroy and Goss[3] reported the formation of B_1, riboflavin, B_6, and pantothenic acid in the rumen of sheep, and Pearson, Schmidt and Mackey[4] claim that lambs will grow normally on a diet low in nicotinic acid. It is thus difficult to measure the actual need of these animals for these factors. It is known, however, that dogs[5] and pigs[6] require nicotinic acid, and there is no reason to believe that the other higher mammals do not. It also has been shown that the dog, chick and pig require riboflavin[7, 8, 9] and B_6,[10, 11, 12] and pantothenic acid is needed by the chick.[13] Further than this, knowledge concerning the requirements of the various farm animals for the other components of the B group is incomplete.

VITAMIN C

Aside from most of the primates and guinea pigs, vitamin C is apparently nonessential because it presumably can be synthesized in the body of the other animals. It is generally assumed that all of the higher animals, including fowls, need the antirachitic factor as vitamin D in the diet or as direct irradiation with sunlight or other source of ultraviolet light.

VITAMIN E

It has never been demonstrated that any of the higher mammals need vitamin E.*

*From the department of physiological chemistry, School of Medicine, University of Pennsylvania; presented at the 40th annual conference of veterinarians at the University of Pennsylvania, January 4-5, 1940.

*Since this paper was first presented, a report has appeared (Anderson, H. D., Elvehjem, C. A., and Gonce, Jr., J. E.: Proc. Soc. Exp. Biol. & Med., xliii, 1939, p. 750), showing that vitamin E is an essential for the dog.

According to Vogt-Moller and Bay, habitual abortions in both cattle[14] and pigs[15] have been successfully treated with vitamin E preparations. On the other hand, Thomas *et al*[16] could not demonstrate that vitamin E is an essential for goats, sheep or rabbits. Chicks, both male and female, definitely need vitamin E.[17]

VITAMIN K

Vitamin K is certainly necessary for the chick[18] and other fowls[19] and is thought to be required by most mammals. However, like B_1, there is an apparent synthesis of this factor in the alimentary canal of some species.[20] Consequently, it is difficult to demonstrate the essential character of this vitamin in these cases.

VITAMINS A AND D MOST LIKELY TO BE DEFICIENT IN DIET OF FARM ANIMALS

Which of these vitamins that are essential are most likely to be deficient in the diet of farm animals? Nearly all of the B components, with the exception of nicotinic acid, are found in liberal amounts in grains and to some extent in roughages. They are also quite stable. It is thus not surprising that the rations of most farm animals are seldom lacking in these essentials. Since Arnold and Elvehjem[21] recently reviewed the subject of the relation of the vitamin B complex to the nutrition of domestic animals, it is unnecessary to consider these factors further. Vitamin E is found in abundance in whole grains, and vitamin K in roughages. They are, therefore, seldom deficient in the average rations used on the farm. However, the same can not be said of vitamins A and D.

Most of the domestic animals are herbivorous and depend upon the plant kingdom for their supply of vitamin A. In the plant, vitamin A is not present as such, that is, in the same form in which it functions in the animal body. Instead, plants furnish vitamin A to the animal in the form of yellow pigments, of which there are four. The animals have the ability to convert these pigments into colorless vitamin A. Three of these pigments are carotenes and designated as alpha, beta and gamma carotene. In addition to the carotenes the closely related pigment, cryptoxanthin, has vitamin A activity. In the conversion of these pigments to vitamin A it has been shown that one molecule of beta carotene can be converted into two molecules of vitamin A, whereas but one molecule of the vitamin is produced from each molecule of the other pigments mentioned. Thus, for a given weight of pigment, beta carotene has approximately twice the vitamin A activity of the others.

In attempting to determine if a given plant or food contains these pigments, it should not be judged solely on its color, as there are several similar-appearing plant pigments which have no vitamin A activity. It is known, however, that yellow corn contains significant amounts of cryptoxanthin,[22] but it is the only commonly used grain that can be considered as a moderately good source of this dietary essential. By far the best and most reliable source of vitamin A is fresh green grass, followed in importance by properly cured leafy hays, such as alfalfa and clover. Practically all green, rapidly growing, leafy plant material contains an abundance of carotene. Carotene, however, is not a very stable substance and a considerable portion of it is destroyed during drying.[23] Green color in this case is a fairly reliable index as to whether or not vitamin A activity has been destroyed. Chlorophyl, like carotene, is an unstable compound and is destroyed rather easily during the process of curing. If the forage has been handled in such a manner as to retain its green color, it is safe to assume that an appreciable amount of the carotene has been preserved. However, under conditions of storage carotene seems to be destroyed more readily than chlorophyl.[24] Meigs and Converse[25] showed that young, green, rapidly growing alfalfa may contain ten times as much vitamin A activity as a No. 1 grade alfalfa hay, and the first grade alfalfa may contain 30 times the vitamin A activity of a third grade timothy hay.

With these facts in mind it is not difficult to predict with some degree of accuracy when a deficiency of vitamin A

might be expected. The animal body has a great power for storing this factor. Consequently, if an animal, such as a dairy cow, is on green pasture all summer, there is little danger that a vitamin A deficiency will develop during the winter, even though the winter feed may contain an insufficient amount. If the cow is giving milk during the winter months, there is a rapid decrease in the amount of vitamin A which is secreted in the milk, unless liberal quantities are supplied in the diet during this period. In fact, at the Texas Agricultural Experiment Station, Fraps, Copeland and Freichler[26] found that it was impossible to maintain the summer level in the milk even though they fed as much as 116,000 I. U. of vitamin A daily. In certain localities where drought is rather common and the pastures tend to dry up, there is actually danger that dairy cattle will develop a deficiency. It was reported by Hart, Mead and Guilbert[27] in California that range-fed cattle may at times show typical symptoms of vitamin A lack. In these cases the cows either failed to reproduce or the calves were born dead or very weak, and in some cases the calves showed typical eye symptoms. Retention of the placenta was also common, and in some cases the cows gave evidence of night blindness. Somewhat similar observations[28] were made in Kansas during the drouth of a few years ago. Special care must be given cows which are kept indoors the year around. Meigs[25] reported that cows which were fed for some time with overripe timothy hay as the only roughage failed to reproduce.

VITAMIN A DEFICIENCY MORE PREVALENT IN CALVES THAN IN COWS

A deficiency of vitamin A among calves probably occurs much more frequently and over a greater area than it does in cows. A rather common practice is to feed calves during the first summer on skim milk, which contains very little vitamin A, and some kind of a calf-feed concentrate. During this period there may be an actual deficiency resulting in growth and appearance below normal. At best, the young animal has little opportunity to store away extra vitamin A for the winter months. To make matters worse, the young dry stock are frequently given a hay of inferior grade, the better grades being saved for the cows which are producing milk. Meigs and Converse[25] believe that special precautions should be taken from birth until the animals are at least 6 months of age.

PIGS ARE HIGHLY SUSCEPTIBLE TO VITAMIN A DEFICIENCY

What has been said concerning the raising of calves applies to an even greater extent to pigs, for, generally, green pasture forms a less important part of the diet of hogs than of cattle. Dunlop[29] has gone so far as to state that most diets used for pigs are deficient in vitamin A. He also feels that it is not desirable to supply the needed amount in the form of yellow corn and alfalfa. He recommends the use of concentrates.

In 1923, Emmett and Peacock[30] definitely showed that the chick requires vitamin A. Repeated observations, both experimental and practical, have emphasized the importance of an abundant supply of this factor in the rations of chicks, especially laying hens. Sherwood and Fraps[31] stated that they believe that ordinary poultry rations do not contain sufficient vitamin A for heavy egg production, and Gish and Payne[32] have just published a report on the importance of green material in poultry management. Tepper and Durgin[33] and Almquist and Mecchi[34] also reported recently on the vitamin A requirement of growing chicks and laying hens.

VITAMIN D

Another vitamin which is quite likely to be deficient in the diet of farm animals is vitamin D. Few of the foods which are commonly given to either humans or domestic animals contain appreciable amounts of the antirachitic factor. Apparently, nature intended that we should get our supply of this essential directly from the sun, which can convert inactive provitamin D into the active form. Since this is the case, it follows that any animal,

especially if it be a young, growing animal, which is deprived of direct sunlight for any great length of time will be deficient in this factor. As in the case of vitamin A, considerable work has been done relative to the needs of calves for vitamin D. Without it, either in the food or as direct sunlight, these animals will develop characteristic symptoms of rickets: Poor growth, rough coat, bowlegs, enlarged joints and low serum calcium and phosphorus.[35] Rupel, Bohstedt and Hart[35] at Wisconsin reported that a condition of mild rickets was common in calves on the farms of that state. Similar observations were reported from Minnesota by Gullickson, Palmer and Boyd,[36] and Hastings[37] in Illinois has reported the occurrence of tetany in calves which were kept indoors.

In the case of very young calves which are confined inside and which have not yet started consuming hay, it is best to give some form of concentrate. For older animals, aside from direct sunlight, again we depend upon roughage for vitamin D. Unfortunately, the active form of the vitamin is formed during the curing process, which means that methods which lead to the conservation of large quantities of vitamin A prevent the activation of vitamin D.[23] As long as the plant is green and growing, the inactive form of vitamin D is not converted to the active form. However, as soon as the plant is cut and drying starts, conversion from the inactive to the active form begins to take place, provided the plant material is exposed to the sun during the curing process. Here again, the leafy legume hays apparently can be activated to a greater extent than other types of roughage,[38] although it has been reported that other roughages[39] contain appreciable quantities of this factor. Thus, it appears that a good grade of alfalfa that is cut while still green and dried carefully in the sun provides liberal amounts of vitamin D while still retaining a considerable portion of its vitamin A.[23]

Again, as with vitamin A, young, growing pigs are very susceptible to a lack of vitamin D. If direct sunlight is not available, then some source of the antirachitic factor must be supplied in the diet. What was said in regard to the need of the chick for vitamin A can be repeated with increased emphasis concerning the need of this species for vitamin D. It is common knowledge that it is impossible to raise chicks indoors without giving vitamin D in some form. In this connection there is one important point which must be considered. Chemically there are several different compounds which can be activated, that is, converted into vitamin D, by ultraviolet light. The two best known, and probably most important and most widely distributed, are ergosterol and 7-dehydrocholesterol. The former is of plant origin and the latter of animal origin. In the pure, active form these compounds are approximately equal in their antirachitic activity for the rat. That is, 1 mg. of each contains about 40,000 I. U.[40]

It was recognized early that irradiated ergosterol is very ineffective in the prevention of leg weakness in chicks.[41] On a rat-unit basis, about 50 times as much irradiated ergosterol is required as cod liver oil.[40] Present evidence indicates[42] that the principal antirachitic substance in cod liver oil is the active form of 7-dehydrocholesterol. This form of vitamin D is efficacious in protecting chicks against leg weakness or rickets.[43] The accepted method of standardizing antirachitic substances is to use the rat. As can be seen, such a method is practically useless when one attempts to determine the potency of antirachitic agents to be used with chicks. For, if the preparation contains irradiated ergosterol, it might show a high antirachitic value by the rat-assay method, and at the same time it would be practically useless as an antirachitic for the chick. Thus, the only accepted method for determining the antirachitic activity of poultry feeds is the use of the chick as the test animal.

What has been said about the chick is undoubtedly true of the other domestic fowls. Since the vitamin D of forage is of plant origin and, hence, is activated ergosterol or a similar compound, it is ineffective for the chick. On the other hand, all experiments, to date, indicate that the

domestic animals of the mammal class respond more like the rat to the various forms of vitamin D. This means that not only can stock feeds be assayed for their vitamin D activity on the rat but, of greater importance, irradiated plant materials, such as hays, are effective antirachitic agents for this class of animals.

FISH LIVER OILS AND YEAST AS SOURCES OF VITAMIN D

At times when it is impossible to supply the necessary factors as natural foods or direct sunlight, it becomes necessary to use special vitamin products. In the use of cod liver oil it must be borne in mind that this substance is definitely toxic to some animals, especially the herbivora.[44] However, Davis and Maynard[45] have reported that calves can withstand cod liver oil better than other herbivora, and probably sufficient vitamin D can be given by this method. Nestler[46] found that 8 per cent cod liver oil is definitely toxic to the laying hen but that 2 per cent is well tolerated. It also should be remembered that cod liver oil becomes rancid quickly, especially when exposed to the air, as in a dry feed mixture. Furthermore, under similar conditions vitamin A is readily destroyed by oxidation.[47]

Experiments conducted at the Pennsylvania State College[48] indicated that irradiated yeast is a reliable and inexpensive source of vitamin D for calves. It is possible that vitamin D can be conveniently supplied to other mammals in this form. However, the provitamin of yeast is ergosterol and, consequently, is a poor source of the antirachitic factor for chicks and other fowls. In choosing between cod (or other fish) liver oil and irradiated yeast as a source of vitamin D, one should remember that the latter contains little or no vitamin A, whereas the former is one of the best sources of this dietary essential.

SUMMARY

Vitamins A and D are more likely to be deficient in the diets of ordinary farm animals than any of the other vitamins. Both of these dietary essentials can be best supplied by feeding liberal quantities of carefully sun-cured, leafy hays which are cut before becoming too ripe. The vitamin D of roughages is not effective for poultry and some form of concentrate, especially fish liver oils, should be used. In the use of cod liver oil it must be kept in mind that it is toxic for certain animals; it becomes rancid easily, and vitamin A, which it contains, is rapidly destroyed when the oil is mixed with dry feeds.

References

[1]Frost, D. V., and Elvehjem, C. A.: Jour. Biol. Chem., cxxi (1937), p. 822.
[2]Almquist, H. J., and Mecchi, E.: Jour. Biol. Chem., cxxvi (1938), p. 407.
[3]McElroy, L. W., and Goss, H.: Jour. Biol. Chem., cxxx (1939), p. 437.
[4]Pearson, P. B., Schmidt, H., and Mackey, A. K.: Proc. Soc. Exp. Biol. & Med., xl (1939), p. 423.
[5]Elvehjem, C. A., Madden, R. J., Strong, F. M., and Woolley, D. W.: Jour. Biol. Chem., cxxiii (1938), p. 137.
[6]Chick, H., Macrae, T. F., Martin, A. J. P., and Martin, C. J.: Biochem. Jour., xxxii (1938), p. 10.
[7]Sebrell W. H., and Onstott, R. H.: U. S. Pub. Health Rpts., liii (1938), p. 83.
[8]Lepkovsky, S., and Jukes, T. H.: Jour. Nutri., xii (1936), p. 515.
[9]Hughes, E. H.: Jour. Nutri., xvii (1939), p. 527.
[10]Fouts, P. J., Helmer, O. M., Lepkovsky, S., and Jukes, T. H.: Jour. Nutri., xvi (1938), p. 197.
[11]Hegsted, D. M., Oleson, J. J., Elvehjem, C. A., and Hart, E. B.: Jour. Biol. Chem., cxxx (1939), p. 423.
[12]Chick, H., Macrae, T. F., Martin, A. J. P., and Martin, C. J.: Biochem. Jour., xxxii (1938), p. 2207.
[13]Jukes, T. H.: Jour. Amer. Chem. Soc., lxi (1939), p. 975. Woolley, D. W., Waisman, H. A., and Elvehjem, C. A.: Jour. Amer. Chem. Soc., lxi (1939), p. 977.
[14]Vogt-Moller, P., and Bay, F.: Vet. Jour., lxxxvii (1931), p. 165.
[15]Bay, F., and Vogt-Moller, P.: Vet. Jour., xc (1934), p. 288.
[16]Thomas, B. H., Cannon, C. Y., McNutt, S. H., and Underbjerg, G.: Jour Nutri., xv (1938), p. 10.
[17]Adamstone, F. B.: Jour. Morph., lii (1931), p. 47. Adamstone, F. B., and Card, L. E.: Jour. Morph., lvi (1934), p. 339.
[18]Dam, H.: Biochem. Jour., xxix (1935), p. 1273.
[19]Dam, H., Schnheyder, F., and Lewis, L.: Biochem. Jour., xxxi (1937), p. 22.
[20]Dam, H., and Glavind, J.: Lancet, 1 (1938), p. 720.
[21]Arnold, A., and Elvehjem, C. A.: Jour. A.V.M.A., xcvi (Jan. 1940), p. 56.
[22]Kuhn, R., and Grundmann, C.: Ber. deut. Chem. Gesel., lxvii B (1934), p. 593.
[23]Russell, W. C.: Jour. Biol. Chem., lxxxv (1929), p. 289. Smith, M. C., and Briggs, I. A.: Jour. Agr. Res., xlvi (1933), pp. 229 and 235.
[24]Kane, E. A., Weiseman, H. G., and Cary, C. A.: Jour. Agr. Res., lv (1937), p. 837.
[25]Meigs, E. B., and Converse, H. T.: Amer. Soc. Ani. Prod., Proc. 26th Ann. Meet. (1933), p. 58. Chem. Absts., xxviii (1934), col. 5105.
[26]Fraps, G. S., Copeland, O. C., and Freichler, R.: Texas Agr. Exp. Sta. Bul. 495 (1934).
[27]Hart, G. H., and Guilbert, H. R.: California Agr. Exp. Sta. Bul. 560 (1933).
[28]Hughes, J. S.: No. Amer. Vet., xvii (1936), 3, p. 22.
[29]Dunlop, G.: Jour. Agr. Sci., xxv (1935), p. 217.

[30]Emmett, A. D., and Peacock, G.: Jour. Biol. Chem., lvi (1923), p. 679.
[31]Sherwood, R. M., and Fraps, G. S.: Texas Agr. Exp. Sta. Bul. 493 (1934).
[32]Gish, C. L., and Payne, L. F.: Poultry Sci., xix (1940), p. 35.
[33]Tepper, A. E., and Durgin, R. C.: New Hampshire Agr. Exp. Sta. Bul. 310 (1938).
[34]Almquist, H. J., and Mecchi, E.: Poultry Sci., xviii (1939), p. 129.
[35]Rupel, I. W., Bohstedt, G., and Hart, E. B.: Wisconsin Exp. Sta. Res. Bul. 115 (1933).
[36]Gullickson, T. W., Palmer, L. S., and Boyd, W. L.: Minnesota Agr. Exp. Sta. Tech. Bul. 105 (1935).
[37]Hastings, C. C.: No. Amer. Vet., xx (1939), 4, p. 47.
[38]Wallis, G. C.: Jour. Agr. Res., lvii (1938), p. 393.
[39]Bechtel, H. E., Huffman, C. F., Duncan, C. W., and Hoppert, C. A.: Jour. Dairy Sci., xix (1936), p. 359. Huffman, C. F., and Duncan, C. W.: Jour. Dairy Sci., xviii (1935), p. 511.
[40]Remp, D. G., and Marshall, I. H.: Jour. Nutri., xv (1938), p. 525.
[41]Hess, A. F., and Supplee, G. C.: Proc. Soc. Exp. Biol. & Med., xxvii (1929-30), p. 609.
[42]Bills, C. E.: Jour. Amer. Med. Assn., cx (1938), p. 2150.
[43]Waddell, J.: Jour. Biol. Chem., cv (1934), p. 711. Bethke, R. M., Record, P. R., Wilder, O. H. M., and Kennard, D. C.: Poultry Sci., xvi (1937), p. 438.
[44]Madsen, L. L., McCay, C. M., and Maynard, L. A.: Cornell Univ. Agr. Exp. Sta. Memoir 178 (1935).
[45]Davis, G. K., and Maynard, L. A.: Jour. Dairy Sci., xxi (1938), p. 143.
[46]Nestler, R. B.: Jour. Agr. Res., lvi (1938), p. 209.
[47]Jones, J. H.: Jour. Biol. Chem., lxxv (1927), p. 139.
[48]Bechdel, S. I., Hilston, N. W., Guerrant, N. B., and Dutcher, R. A.: Pennsylvania State Coll. Exp. Sta. Bul. 364 (1938).

Hydrogen Peroxide Lavage in Helminthiasis of Dogs

Whitney's intestinal lavage with a solution of hydrogen peroxide was given a high rating* as a means of ridding the digestive tract of its helminthic inhabitants: Ascarids, hookworms, tapeworms. All of these yield to the deadly action of this simple remedy.

After evacuating the rectum of fecal material, a dilution of hydrogen peroxide, 1 per cent, is instilled in the usual manner until a through and through lavage is accomplished. Peroxide of hydrogen at this strength is a vermicide. The worms washed out are dead or fatally injured. Subsequent treatment is rarely required. The 2 per cent solution formerly recommended is believed to be too irritant and unnecessary.

*E. J. Frick before the Kansas State Veterinary Medical Association, 1940.

The Adrenal Glands

The adrenal glands, or suprarenal capsules as they are also named, because they fit like a hood on the cephalic end of the kidneys, were discovered by Eustachi in 1543, but little attention was paid to them until Addison, in 1856, incriminated dysfunction of them as the cause of the fatal human disease (now identified as tuberculous) that has since borne his name.

Important facts about these mysterious bodies were added by Brown-Sequard (1891), Oliver and Shaffer (1895), and Takamine and Aldrich (1901). It was, however, only in 1927, when the two American biologists, Stewart and Rogoff, succeeded in isolating the active principle from the cortex of the glands, and Reichstein (1936) crystallized it, that the present knowledge of them became more clear. The crystallized product is the specific hormone of the cortex. It maintains life in subjects from which the glands are removed.

The physiology of the cortex and of the medulla are entirely different studies, and the chemical formulas of the two derivatives used medicinally—cortin and epinephrine—also are different.

The pathology of the adrenal glands is of no special importance in veterinary medicine. No one has yet demonstrated the presence of a disease of them or attributable to them. Only vague and unproved theories as to their relation to certain physiological aberrations, such as pseudo-hermaphroditism and male plumage on female birds, are mentioned as being of suprarenal origin. Otherwise, the use of epinephrine as a vasoconstrictor in local anesthesia and as an antianaphylactic agent is the clinician's only interest in these vital organs.

It is commonly thought that dyes are obtained from coal tar. The truth is that coal tar furnishes the raw material for dyes in the form of essential compounds from which dyes and other important materials are built up step by step.—*Science Digest.*

Phenothiazine as an Anthelmintic for the Removal of Intestinal Worms from Swine*

By LEONARD E. SWANSON, B.Sc., D.V.M., PAUL D. HARWOOD,† Ph.D., and JAMES W. CONNELLY

PRELIMINARY tests with phenothiazine, suggesting that this drug might prove to be valuable as an anthelmintic for the removal of worms from swine, were described by Harwood, Jerstad and Swanson.[7] The limited amount of information available at that time suggested also that phenothiazine might have a margin of safety sufficiently wide to warrant its administration with the feed. Accordingly, a number of experiments were performed to determine the anthelmintic efficacy of phenothiazine for the removal of gastrointestinal parasites from swine. This study is by no means complete, but sufficient progress has been made to warrant presentation of the available data.

REVIEW OF LITERATURE

Phenothiazine, also called thiodiphenylamine, is a snythetic, heterocylic, organic compound that was first synthesized in the 19th century. It remained a chemical curiosity until recently, when investigators of the U. S. bureau of entomology and plant quarantine demonstrated that it possessed promise as an insecticide.[13] That this chemical was also an anthelmintic of promise for swine was first shown by Harwood, Jerstad and Swanson.[7] Later, Harwood, Habermann and Jerstad,[5] Swales,[14] Gordon,[1] Gordon and Whitten,[2] Roberts,[11] and Habermann and Harwood[3] reported promising results with phenothiazine as an anthelmintic for the removal of worms from sheep. McNaught, Beard and DeEds[8] reported that phenothiazine in the diet affects adversely the number of trichinae which develop in experimentally infested rats.

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†The experiments to determine the efficacy of phenothiazine were outlined by Paul D. Harwood, who also prepared the data and discussion presented in this paper. The experiments were carried out at Moultrie, Ga., by Leonard E. Swanson and James W. Connelly.

MATERIALS AND METHODS

The phenothiazine used in the earlier experiments was obtained from commercial sources and, unknown to the writers, a conditioning agent had been added to the chemical in order to make the product suitable for use as an insecticide. In later tests the recrystallized product was used, the conditioning agent having been removed by Harry D. Anspon of the University of Maryland. To purify the chemical, the conditioned product was dissolved in hot toluene, recrystallized, and separated by filtration from the solvent.

Cull pigs of mixed breeds were used in the experiments given in this paper. The pigs were purchased locally and confined in individual cages. After a fasting period of 18 to 24 hours, the pigs were given conditioned phenothiazine at dose rates varying from .25 to 0.9 Gm. per pound of body weight. The dose was administered either mixed with the feed or in hard gelatin capsules. The same procedure was followed with recrystallized phenothiazine, except that the dose was reduced to .05 to 0.2 Gm. per pound of body weight, and that all doses were administered to the animals in hard gelatin capsules. After treatment, the feces eliminated by each pig were collected daily, examined by the usual technic, and any worms present were counted.

Approximately one week after treatment the animals were killed and all worms not removed by the treatment were collected, identified and counted. In some instances, examination of feces after treatment for worm eggs indicated that the host animals were still infested with ascarids; such pigs were redosed with phenothiazine as indicated in the tables. Each administration of the drug was considered to be an experiment, and the worms which were not eliminated following treatment are recorded separately in the tables for each experi-

TABLE I—Efficacy of conditioned phenothiazine* for the removal of ascarids and nodular worms from swine.

Designation and Sex of Pig	Weight (Kg.)	Dose of Drug (Gm.)	Worms Recovered from Feces			Days Between Treatment and Necropsy	Worms Not Eliminated Following Treatment		
			Immature Ascarids	Mature Ascarids	Nodular Worms		Immature Ascarids	Mature Ascarids	Nodular Worms
3 ♀	4.0	4.5	0	84	61	7	4	0	1
4 ♂	4.5	4.8	0	3	3	5	0	5	0
5 ♀	3.2	3.5	0	5	122	8	0	5	9
6 ♀	9.0	10.0	0	0	1,136	8	0	0	25
9 ♂	3.9	4.3	0	0	4	7	0	5	1
10 ♀	25.0	27.5	0	0	25	11	2	0	27
12 ♀	8.4	9.3	0	0	1,149	9	0	0	53
13 ♀	10.0	12.0	0	0	120	9	1	0	3
14 ♀	6.4	7.0	0	4	706	10	0	4	48
15 ♂	5.8	6.4	0	80	352	10	0	6	190
16 ♂	13.2	14.5	0	6	788	9	0	3	3
17 ♀	12.8	14.0	0	0	88	9	0	3	8
19 ♂	17.8	20.0	0	11	11	9	0	1	0
20 ♂	26.8	50.0	0	0	11	10	0	2	0
21 ♂ †	12.3	16.9	0	1	0	†	0	12	0
21 ♂	12.3	16.9	0	0	0	7	0	12	0
22 ♀ †	12.0	18.6	0	6	0	†	0	26	0
22 ♀	12.0	18.6	0	2	0	7	0	24	0
23 ♂	11.8	15.5	0	0	2	8	1	0	0
24 ♂ †	7.6	12.0	0	1	2	†	16	5	0
24 ♂	7.6	12.0	0	0	0	7	16	5	0
25 ♂	8.2	10.8	0	1	0	13	0	3	0
27 ♂	23.3	40.8	0	9	113	9	0	0	1
28 ♂ †	21.9	10.9	0	1	20	†	2	6	40
28 ♂	18.2	10.9	2	1	40	6	0	5	0

*The dose was administered to pig 5 as an electuary made with molasses; to pigs 4 and 28 in hard gelatin capsules; to all other animals in a mixture of ground feeds which the animals ate readily.

†To obtain evidence on the effects of repeated doses of phenothiazine, this animal was not necropsied at the usual time, but redosed as shown in this table. The number of worms not eliminated following the first treatment was calculated from the data obtained during the second treatment of the animal involved.

ment. This was possible since all feces eliminated by a pig subsequent to the first treatment were examined for worms, and after the last treatment the pig was necropsied to determine the number of worms remaining.

A few experiments to determine the toxicity of phenothiazine for swine were carried out by one of the writers (P. D. H.), at the Agricultural Research Center, Beltsville, Md.

Experimental Data

Detailed results of 25 experiments involving 22 pigs that received conditioned phenothiazine are presented in table I. The treatment removed 4,753 (92.1 per cent) of the 5,162 nodular worms (Oesophagostomum spp.), as well as 2 (4.5 per cent) of 44 immature ascarids (*Ascaris suis*), and 215 (62.3 per cent) of 345 mature ascarids present in the host animals at the time of treatment. All female ascarids ranging in size from larval worms no bigger than those found in the lungs up to worms about 4 in. long were regarded in this work as immature because they contained no ripe eggs. Males up to about 2½ in. long also were regarded as immature.

Detailed results of 20 tests, involving 14 pigs, that were carried out with recrystallized phenothiazine are presented in table II. Following treatment the pigs receiving the recrystallized chemical eliminated 577 (92.3 per cent) of 625 nodular worms, 48 (19.1 per cent) of 451 immature ascarids and 116 (58.9 per cent) of 197 mature ascarids present in these host animals at the time of treatment.

Apparently, recrystallized phenothiazine in the doses used was as effective as the much larger doses of conditioned phenothiazine. The average dose of the recrystallized product per pig was 4.2 Gm.

(.17 oz.); the average dose of the conditioned chemical was 14.9 Gm. (0.5 oz.). These results are in agreement with those obtained in sheep.[3, 5] The data presented in tables I and II also suggest that phenothiazine is more effective for the removal of mature ascarids when a relatively large number of worms are present than when only a few are present. The average of the per cent efficacies obtained with phenothiazine for the removal of ascarids was 23.6 when each pig was infested with fewer than eight worms, and 51.6 when each pig was infested with eight or more worms. The probability that the difference between these means was due to a chance distribution of factors was determined by ordinary methods of statistical analysis to be less than 1 in 20. Similarly, the difference between the average of the per cent efficacies of the chemical in pigs infested with fewer than eight mature ascarids each and the average in those infested with more than 70 mature ascarids each is 72.7, and for this figure "P" is much less than 0.001.

Twenty-four of the pigs were infested with stomach worms (Ascarops, Physocephalus, and Hyostrongylus) at the time of treatment. Following treatment, a total of 30 stomach worms were eliminated, but 1,000 to 2,000 stomach worms were found at necropsy (the exact number was not determined). Following treatment, ten hookworms (*Crassisoma urosubulatum*) were eliminated from pig 6, the only animal infested with this species of parasite, but 101 hookworms were removed at necropsy of this host animal. Thirty whipworms (*Trichuris trichiura*) were eliminated by 25 pigs which were infested with a total of 250 whipworms prior to treatment. Only five of the pigs, namely, Nos. 3, 4, 5, 6 and 18, used in this investigation were infested with thornyheads. None of the total of 21 thorny-headed worms (*Macracanthorhynchus hirundinaceus*) was removed by the treatment.

In order to obtain information on the possibility of administering phenothiazine to swine in the feed, 40 pigs were given conditioned phenothiazine at the rate of 0.5 Gm. (8 gr.) per pound of body

TABLE II—Efficacy of recrystallized phenothiazine administered in capsule for the removal of ascarids and nodular worms from swine.

Designation and Sex of Pig	Weight (Kg.)	Dose of Drug (Gm.)	Worms Recovered from Feces			Days Between Treatment and Necropsy	Worms Not Eliminated Following Treatment		
			Immature Ascarids	Mature Ascarids	Nodular Worms		Immature Ascarids	Mature Ascarids	Nodular Worms
29 ♀	16.9	3.7	0	5	21	8	1	0	0
30 ♀	30.0	6.5	0	7	50	7	0	1	0
31 ♀	33.2	7.3	0	1	284	7	0	7	0
32 ♂	31.9	10.5	0	1	18	7	0	3	0
33 ♀	27.3	12.0	0	0	140	7	0	0	2
34 ♀	22.8	2.4	0	69	1	7	42	3	0
35 ♂	10.9	2.4	0	0	2	7	93	3	0
36 ♂	10.1	2.3	0	1	4	7	25	0	0
37 ♀*	14.6	3.2	0	11	42	*	0	8	0
37 ♀	14.6	3.2	0	2	0	6	0	6	0
38 ♂*	14.6	2.2	52	5	6	*	98	0	0
38 ♂	14.6	2.2	0	0	0	5	90	0	0
39 ♂*	12.4	1.4	1	0	0	*	10	5	17
39 ♂	12.4	1.4	0	0	0	6	10	5	17
40 ♀*	18.3	4.0	4	0	2	*	41	26	1
40 ♀	23.7	5.2	41	19	1	8	0	6	0
41 ♀*	14.6	3.2	0	0	0	*	0	3	7
41 ♀	18.7	4.1	0	0	5	8	0	3	2
42 ♀*	13.8	3.0	2	0	2	*	1	6	22
42 ♀	17.3	3.8	1	0	5	8	0	6	14

*To obtain evidence on the effects of repeated doses of phenothiazine, this animal was not autopsied at the usual time, but redosed as shown in the table. The number of worms not eliminated following the first treatment was calculated from the data obtained during the second treatment of the animal involved.

weight. Prior to dosing, the pigs were fasted for 24 hours, and the drug was mixed with a ground feed at the rate of 4 lbs. of feed to 1 lb. of phenothiazine. The entire medicated mixture, which was placed in a trough long enough for all animals to feed at one time, was consumed promptly. Unfortunately, it was impossible to gather any reliable information as to the anthelmintic efficacy of the drug in this test. No symptoms of intoxication developed in any of these pigs following treatment, but all eliminated urine which turned red on exposure to air, thus demonstrating that all had partaken of the drug to some extent.

Several experiments were undertaken to determine whether treatment of swine with phenothiazine is likely to lead to serious intoxication of the animals. Three hundred grams (10 oz.) of conditioned phenothiazine mixed with various feeds was given to each of five pigs weighing from 136 to 225 kg. (300 to 495 lbs.) each. A mild enteritis was found in three of the five pigs, but since a similar enteritis was found in one of the control animals, the significance of these findings is doubtful. To each of two pigs weighing 114 and 136 kg. (250 and 300 lbs.), respectively, 300 Gm. of conditioned phenothiazine was administered in 1,200 Gm. (2.5 lbs.) of feed on each of the following dates: November 23, 26 and 30 and December 13, 1938. At necropsy on December 19, 1938, these pigs exhibited moderately severe enteritis, and nephritis, apparently due to the action of the drug.

It may be noted that these pigs ate the drug mixtures with comparatively little hesitation. To one pig weighing 68 kg. (150 lbs.), 30 Gm. (1 oz.) of phenothiazine was administered in hard gelatin capsules in the morning. Immediately afterward, the pig was offered 70 Gm. (2.3 oz.) of phenothiazine mixed with tankage and corn meal. As this dose was consumed readily, 50 Gm. (1.6 oz.) of phenothiazine mixed with tankage and corn meal was offered on the afternoon of the same day and was likewise consumed. Following treatment, the pig exhibited no clinical symptoms and, at necropsy on April 14, 1939, the only pathology detected was a mild enteritis.

COMPARISON OF RESPECTIVE EFFICACIES OF PHENOTHIAZINE AND OIL OF CHENOPODIUM

Oil of chenopodium is generally considered to be the most effective ascaricide for use in swine.[12] Hall and Foster[4] reported that in doses of 1 to 2 cc. per host animal, oil of chenopodium removed all of eleven ascarids present in three pigs at the time of treatment, but the drug removed only 26 of 786 nodular worms present in these test animals. Mote[10] reported the elimination of only seven of a total of 20 ascarids present in four pigs at the time of treatment with oil of chenopodium. Morris and Martin[9] reported that this drug removed three of a total of six ascarids present in 13 pigs at the time of treatment. The latter authors reported evident symptoms of intoxication. None of the authors referred to indicated whether or not the ascarids removed were all mature.

The following unpublished records of experiments performed by the senior author at Moultrie, Ga., with oil of chenopodium as an anthelmintic in swine that had been fasted for 24 hours may be added to the above reports.

Pig 52.—Weight, 27 kg. (60 lbs.); dose, 1.75 cc. (27 minims); no purge; date treated, June 9, 1939; two mature ascarids eliminated. Animal fasted and redosed in the same manner on June 17, 1939; three mature ascarids eliminated. Necropsy findings, June 21, 1939: Three mature ascarids, 406 nodular worms, one whipworm; gastritis present.

Pig 53.—Weight, 12.3 kg. (27 lbs.); dose, 1.08 cc. (16 minims); purge, 20 cc. (0.6 oz.) of castor oil; date treated, June 9, 1939; one mature ascarid eliminated on June 10, 1939. Necropsy findings, June 20, 1939: No ascarids, 155 nodular worms; no pathology that could be connected with the treatment was observed.

Pig 54.—Weight, 3.7 kg. (8 lbs.); dose, 0.36 cc. (6 minims) mixed with 7 cc. (2 dr.) of castor oil; date treated, June 9, 1939; 17 mature ascarids eliminated on June 10 and 11, 1939. Necropsy findings, June 15, 1939: Three mature ascarids, 19 nodular worms; no pathology observed.

Thus, in four experiments performed on

three pigs, 23 (65.7 per cent) of 35 mature ascarids were eliminated following treatment with oil of chenopodium. If these results are added to the results of critical tests published previously, medication with oil of chenopodium has resulted in the elimination of 44 (61.1 per cent) of 72 ascarids. Therefore, it appears doubtful that oil of chenopodium is more effective for the removal of ascarids from swine than phenothiazine. Furthermore, the latter drug possesses several practical advantages, as follows: 1) Phenothiazine seems to be more effective in heavy than in light infestations. This drug should give, therefore, greatest relief to the pigs most severely infested. 2) Phenothiazine is very effective for the removal of nodular worms from swine, while chenopodium is practically without effect. 3) Phenothiazine is apparently less toxic to the host than oil of chenopodium. 4) Phenothiazine may be administered in mixtures of feed.

DISCUSSION

The data presented in this paper indicate that phenothiazine in doses far below those which proved even slightly toxic removed approximately 60 per cent of the mature ascarids present in swine at the time of treatment, but that the drug was much less effective against larval and well-grown but sexually immature ascarids. This difference in effectiveness may be due to several factors, namely: 1) The larval ascarids were in the lungs at the time of treatment and migrated to the intestinal tract during the interval which elapsed between treatment of the host and necropsy. 2) The larval and small worms may have been protected by a coating of mucus. 3) Since the data presented in this paper suggest that phenothiazine is more effective as an anthelmintic in hogs heavily infested with mature ascarids than in those lightly infested, it is possible that the ascaricidal principle of phenothiazine is activated by substances excreted by these worms. Naturally, the total of the excretion products from larval and incompletely developed ascarids is less than the total of similar products from large ascarids and, therefore, the excretion from the small worms may not be sufficient to activate the phenothiazine. The reasons for believing that phenothiazine is not an active anthelmintic but that the active agents are substances derived from this chemical in the intestinal tract of host animals were given elsewhere[3] and need not be repeated here.

Although phenothiazine as an anthelmintic for the removal of worms from swine appears to possess several important advantages over other drugs, it is not advisable to recommend the drug unconditionally at this time. Apparently, conditioned phenothiazine is an inferior product for use as an anthelmintic and either the recrystallized chemical or phenothiazine without a conditioner should be used. Tests with commercial phenothiazine lacking the conditioner are now in progress. On the basis of existing information, only tentative dosages of phenothiazine for use in swine can be proposed.

From table II it may be noted that in 15 tests, doses of less than 5 Gm. (.16 oz.) per pig removed only 91 (53.2 per cent) of 171 nodular worms, while in five experiments in which the dose per animal was more than 5 Gm., the drug removed 493 (99.6 per cent) of 495 nodular worms present at the time of treatment. Therefore, it seems reasonable that the dose for swine should not be less than 5 Gm. of recrystallized phenothiazine per animal. Since the percentage of ascarids removed was not satisfactory in these tests, it is suggested that larger doses should be tested in swine.

It already has been pointed out[6] that the actual dose of an anthelmintic should increase with the size of the animal to be treated, but that the dose rate relative to the weight of the animal should decrease. Accordingly, the following schedule of doses, intended only for experimental use, is proposed:

Weight of Pig	*Size of Dose*
Up to 11.4 kg.	5 Gm.
11.4 to 22.8 kg.	8 Gm.
22.8 to 45.5 kg.	12 Gm.
45.5 to 91.0 kg.	20 Gm.
Over 91 kg.	30 Gm.

These dosages are somewhat below the dose of 25 Gm. (0.8 oz.) which Habermann and Harwood[3] proposed for sheep. Perhaps further experimental work will demonstrate that the dosages for the two animals should correspond more closely.

Phenothiazine may be administered to swine in hard gelatin capsules if the operator is sufficiently skilled to avoid lodging the capsules in the pharyngeal pouches, or it may be administered mixed with the feed. The latter method of administration should be undertaken only when pigs are in a pen with which they are familiar. If a group of several pigs are to be treated at one time, sufficient space at the trough for all animals should be provided. Pigs varying greatly in size should not be treated at one time, and the chemical should not be offered to the animals except when they are sufficiently hungry to consume the medicated food at once. The drug may be administered thoroughly mixed with any ground feed that the pigs are accustomed to eating.

SUMMARY AND CONCLUSIONS

Phenothiazine administered to pigs as an anthelmintic seems to be as effective for the removal of mature ascarids as oil of chenopodium, except in cases where only a few worms are present.

Phenothiazine is the only known medicament that is effective for the removal of nodular worms from swine.

Phenothiazine possesses several practical advantages over previously known anthelmintics, as follows: It has a low toxicity, greater efficacy in heavily infested than in lightly infested animals, ease of administration, and anthelmintic activity against more than one species of worm parasite. On the other hand, its low efficacy in removing ascarids when but few worms are present is a decided disadvantage.

Recrystallized phenothiazine is more effective as an anthelmintic than conditioned phenothiazine, which has been proposed for use as an insecticide.

Although further investigations of phenothiazine as an anthelmintic for use in swine are necessary, a table of dosages intended for experimental use is given.

References

[1]Gordon, H. McL.: A preliminary note on the chemotherapy of oesophagostomiasis of sheep with special reference to the efficiency of phenothiazine. Jour. Counc. Sci. & Ind. Res., Australia, xii (1939), 3, pp. 203-206.

[2]Gordon, H. McL., and Whitten, L. K.: A preliminary note on the anthelmintic efficacy of phenothiazine against *Haemonchus contortus* in sheep. 1939.

[3]Habermann, R. T., and Harwood, P. D.: The efficacy of recrystallized phenothiazine for the removal of nematodes from the gastro-intestinal tract of sheep. Vet. Med. (in press).

[4]Hall, M. C., and Foster, W. D.: Efficacy of some anthelmintics. Jour. Agr. Res., xii (1918), 7, pp. 397-447.

[5]Harwood, P. D., Habermann, R. T., and Jerstad, A. C.: Efficacy of commercial phenothiazine in the removal of roundworms from sheep. Vet. Med., xxxiv (1939), 7, pp. 440-443.

[6]Harwood, P. D., Jerstad, A. C., and Underwood, P. C.: The efficacy of n-butyl chloride for the removal of intestinal nematodes, especially whipworms, from dogs. No. Amer. Vet. (in press).

[7]Harwood, P. D., Jerstad, A. C., and Swanson, L. E.: The efficacy of phenothiazine for the removal of ascarids and nodular worms from swine. Jour. Parasitol., xxiv (1938), 6, suppl. pp. 16-17.

[8]McNaught, J. B., Beard, R. R., and DeEds, F.: Effects of sulfanilamide, phenothiazine and thianol in experimental trichinosis. Proc. Soc. Exp. Biol. & Med., xli (1939), 1, pp. 17-20.

[9]Morris, H., and Martin, J. A.: Santonin as an anthelmintic for swine. Jour. A.V.M.A., lxxviii (1931), n. s. 31 (4), pp. 531-538.

[10]Mote, D. C.: Anthelmintic experiments with hogs. Ohio Agr. Exp. Sta. Bul. 378 (1924), pp. 153-182.

[11]Roberts, F. H. S.: A preliminary note on the treatment of oesophagostomiasis in sheep with phenothiazine. Jour. Counc. Sci. & Ind. Res., Australia, xii (1939), 3, p. 208.

[12]Schwartz, B.: Internal parasites of swine. Farmers' Bul. 1787, U. S. Dept. Agr. (1937), 46 pp.

[13]Smith, L. E.: The use of phenothiazine as an insecticide. U. S. Bureau of Entomology and Plant Quarantine, Circular E-399 (1937).

[14]Swales, W. E.: Tests of phenothiazine, a highly efficient anthelmintic. Can. Jour. Comp. Med., iii (1939), 7, pp. 188-194.

The mouths of animals are normally cleaner than those of the human being. Their saliva shows a lower bacterial count than the saliva of man. Dogs, cats, horses, pigs, elephants, baboons, lions and other animals were used to make the comparisons. —*Rosenthal, McNabb and Snyder in the Journal of the American Dental Association.*

Listerella Infection in Swine*

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DURING the past 20 years, a number of conditions occurring in swine, and not previously diagnosed, have been described in the literature. Prior to these descriptions and the recognition of the etiological agents, these conditions were sometimes confused with other diseases. They were frequently grouped under certain disease complexes, while in other instances they remained undiagnosed.

The diagnostician is still confronted with a formidable number of pathological conditions which are not understood and whose causal factors have not been determined up to the present time. The clinical manifestations produced by impactions, food intoxications, certain nutritional deficiencies and infections of the central nervous system are sometimes quite similar, especially during the later stages. The conventional bacteriological examinations and animal-inoculation tests are usually negative in these cases. In certain forms of encephalomyelitis, it is only when material from the central nervous system is injected intracerebrally into susceptible hosts that the conditions can be reproduced. In routine diagnostic procedures, this technic and microscopic examination of the central nervous system are not carried out as frequently as they should be.

In 1926, Murray, Webb and Swan[1] were the first workers to recover a Gram-positive rod from rabbits and guinea pigs which showed generalized infections with hepatic necrosis. In addition to the liver changes and the presence of an exudate in the serous cavities, a marked mononucleosis was observed. Because of this great increase in mononuclear cells, the organism was designated *Bacterium monocytogenes*. No reference was made to changes in the central nervous system. In 1927, Pirie,[2] working in South Africa, isolated a similar organism from rodents. Subsequently, Bergey[3] created the genus Listerella, of which the type species is designated *Listerella monocytogenes* (Murray, *et al*). Since they first described the type species, various workers have found Listerella in sheep,[4,5,6] cattle,[7] humans,[8,9] chickens,[10] and the foxes.[11] The findings, with reference to sheep and cattle listerellosis, indicate that the members of this genus are of considerable importance in veterinary medicine, and the increasing number of human cases being reported suggests that this disease may become a public health problem. Although generalized gross changes are observed in children, chickens and rodents, these are absent in cattle and sheep infected with Listerella. In sheep, Listerella infection is manifested chiefly as a neurotropic infection, the organism being recovered only from the central nervous system to date. Also of great importance is the fact

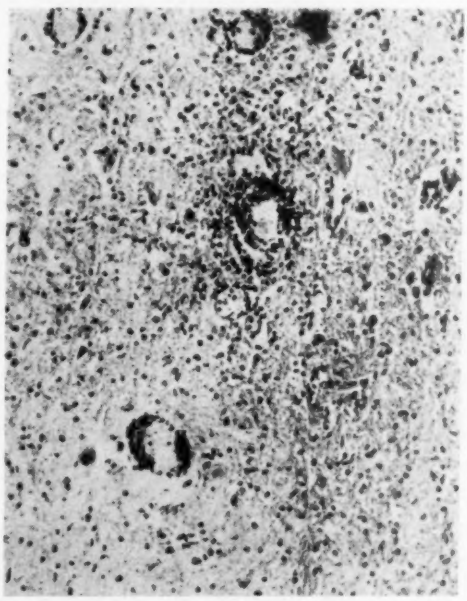

Fig. 1. Perivascular infiltration and necrosis in the brain of a pig (x 130).

*From the Veterinary Research Institute, Iowa State College.

that the cellular changes in the central nervous system are quite similar to the changes associated with some virus infections. Graham *et al*[12] recorded the presence of Listerella in an aborted bovine fetus.

In several localities in Iowa, a number of herds of swine presented symptoms of a central nervous disorder. In one herd, small suckling pigs were involved, while in other herds the pigs weighed 18 to 68 kg. (40 to 150 lbs.). The larger animals trembled. Some individuals dragged the hind legs or showed varying degrees of incoördination while the movements of the forelegs were characterized by an accentuated stilted gait similar to that observed in tetanus. The losses varied in these herds. The highest mortality occurred in the young suckling pigs. Some cases which manifested severe clinical symptoms recovered.

PATHOLOGY

Postmortem examination showed no gross changes that were suggestive of the nature of the disease. Histopathological studies of the central nervous system revealed a marked meningitis characterized by a severe monocytic infiltration. Many blood vessels, especially those in the region of the pons, showed perivascular cuffings (fig. 1). In addition, many foci of monocytic infiltration were found. In some fields a considerable number of polynuclear cells were present. The monocytes in the circulating blood were increased.

BACTERIOLOGY

The direct inoculation of brain material from field cases on culture mediums resulted in the isolation of Listerella bacteria. The organism is Gram positive and about $1.8\mu \times 8\mu$ in size (fig. 2). The morphology, arrangement and behavior of the organisms isolated from swine are similar to those of strains isolated from sheep and cattle.

ANIMAL INOCULATIONS

Brain emulsions made from the original field material were inoculated intracerebrally into rabbits, which died within 24 hours. No difficulty was experienced in recovering the organism from the intracerebrally inoculated rabbits. Numerous characteristic colonies developed on beef-liver-infusion agar following direct inoculations made from the brains of these rabbits.

A single intravenous inoculation of 0.5 cc. (8 minims) of saline suspension (density No. 2 tube, McFarland nephelometer) of Listerella (var. suis) into rabbits failed to cause death. When these rabbits were killed subsequently, microscopic lesions were not found in the brains. However, the rabbits which received intracerebral inoculations of 0.2 cc. (3 minims) of pure culture of Listerella of swine origin died about 24 hours following inoculation. From the brains of these rabbits Listerella was again recovered. Histopathological examination revealed a severe monocytic infiltrative meningitis with early perivascular cuffing in the brain.

EXPERIMENTAL INFECTION OF SWINE

Two pigs weighing approximately 23 kg. (50 lbs.) received intracerebral inoculations of a pure culture of Listerella of swine origin. Both pigs succumbed after about 24 hours. Pure cultures were recovered from the brains of both pigs. Bacteriological examinations made of the parenchymatous organs were negative. Histopathological studies of the brains revealed a severe monocytic infiltrative meningitis, perivascular cuffings and focal infiltrations of monocytes. The most advanced cellular changes were encountered in the tissues surrounding the ventricles and pons, although characteristic changes were found also in other parts of the brain and spinal cord. The lining cells of the ventricles and neural canal were destroyed in many places and monocytic infiltrations appeared in the deeper structures.

The kidneys were slightly swollen, but they presented no specific diagnostic gross changes. Histopathological examination revealed focal monocytic infiltrations which frequently involved the glomeruli and adjoining tissue.

The liver was likewise swollen. It did nôt present a yellow color as is sometimes observed in field cases of ovine listerellosis in which the disease extended over a longer

period. Histopathological examination showed considerable congestion and marked monocytic infiltration.

INTRAMUSCULAR, SUBCUTANEOUS AND ORAL INTRODUCTIONS IN SWINE

Two pigs, each weighing about 55 kg. (120 lbs.), received repeated intramuscular injections of a pure culture of Listerella (var. suis), but the animals failed to develop clinical symptoms. The observations were continued for a period of two months. In previous studies, in which Listerella cultures of ovine origin were used, repeated intramuscular injections into pigs produced no reactions for the duration of a month while the inoculations were being made. About one month after the injections were discontinued, both pigs developed symptoms of a central nervous disorder. The day following the appearance of clinical symptoms these pigs were unable to stand. They were destroyed at this time.

The brains of both pigs showed advanced typical perivascular, focal and meningeal infiltrations, and the organisms were demonstrated in the brain lesions. It should be noted, however, that the pigs which received the ovine cultures weighed only about 25 kg. (55 lbs.) at the close of the experiment, whereas the pigs which received the culture of swine origin each weighed about 68 kg. (150 lbs.) when the period of observation was terminated. The different results obtained might be explained on the basis of age resistance. Among field cases the greatest losses occurred in young suckling pigs. Furthermore, the porcine and ovine strains of Listerella manifested no differences in their effects upon rabbits.

Pure cultures of a porcine strain of Listerella failed to produce visible reactions in pigs weighing about 73 kg. (160 lbs.) when fed by stomach tube. A limited number of intravenous injections in large swine likewise failed to produce untoward effects.

DISCUSSION

A Gram-positive organism identified as a variety of the genus Listerella was isolated from cases of porcine listerellosis characterized by an encephalitis. The presence of hog-cholera virus was eliminated by the inoculation of blood and parenchymatous organs into two series of susceptible pigs. Listerella infection in swine is characterized by varying degrees of incoördination, trembling, a peculiar accentuated "stilted" gait of the forelegs. The temperature may be elevated. In small suckling pigs the incoördination associated with general weakness does not present the picture of a specific disease. These clinical manifestations may be present in other infections.

Listerella encephalitis of swine is distinct from the clinically recognized field condition designated "shivering pigs." The latter is generally believed to be of hereditary origin. Mediums inoculated with brain, liver, kidney and spleen from a limited

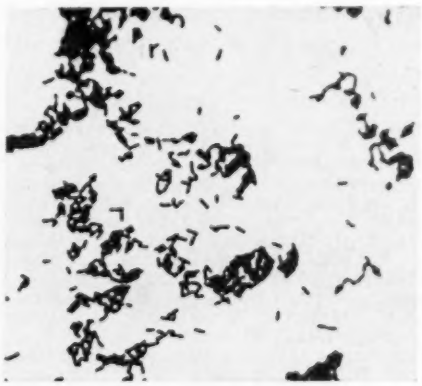

Fig. 2. Listerellae. Hucker's modification of Gram's stain (x 990).

number of so-called shivering pigs failed to show growth. No significant histopathological changes were found in these pigs, whereas in porcine listerellosis the brain presents extensive perivascular infiltrations or cuffings, focal infiltration and severe meningitis. The monocyte is the predominant type of infiltrating cell. However, after necrobiotic changes appear, the infiltrations also may show polynuclear cells.

Listerellosis is of considerable interest from the standpoint of diagnosis and pathology because the cellular reactions produced by the Listerella organism are similar to the changes described in connection with certain virus diseases, especially those of "louping ill" of sheep. The virus of hog

cholera is also capable of producing similar perivascular infiltrations in the central nervous system. In porcine listerellosis no specifically diagnostic gross lesions were found.

Until more is known about this group of infections, sick animals should be isolated and placed in comfortable quarters and nursed to avoid complications. The carcasses of sheep, cattle or swine that succumb to Listerella infections should not be fed to swine, notwithstanding the fact that this was done without untoward results in one severe outbreak among sheep. Listerellosis may prove to be a public health problem. Several cases in man revealed histories of contacts with sick animals, the significance of which was not fully appreciated at the time. In one case, permission was not granted to study the suspected animal.

References

[1]Murray, E. G. D., Webb, R. A. and Swann, M. B. R.: A disease of rabbits characterized by a large mononuclear leucocytosis, caused by a hitherto undescribed bacillus, *Bacterium monocytogenes* (n. sp.). Jour. Path. & Bact., xxix (1926), pp. 407-439.

[2]Pirie, J. H. H. A new disease of veld rodents. "Tiger River disease." So. African Inst. Med. Res., iii (1927), pp. 163-186.

[3]Bergey, D. H.: Manual of Determinative Bacteriology (Williams & Wilkins Co., Baltimore, 1934), pp. 328-329.

[4]Gill, D. A.: Circling disease, a meningo-encephalitis of sheep in New Zealand. Vet. Jour., lxxxix (1933), pp. 258-270.

[5]Jungherr, E.: Ovine encephalomyelitis associated with Listerella infection. Jour. A.V.M.A., xci (1937), n. s. 44 (1), pp. 73-87.

[6]Biester, H. E., and Schwarte, L. H.: Studies on Listerella infection in sheep. Jour. Inf. Dis., lxiv (1939), pp. 135-144.

[7]Jones, F. S., and Little, R. B.: Sporadic encephalitis in cows. Arch. Path., xviii (1934), pp. 580-581.

[8]Schultz, E. W., *et al:* Bacteriological observations of a case of meningo-encephalitis. Proc. Soc. Exp. Biol. & Med., xxxi (1934), pp. 1021-1023.

[9]Burn, C. G.: Clinical and pathological features of an infection caused by a new pathogen of the genus Listerella. Amer. Jour. Path., xii (1936), pp. 341-348.

[10]Seastone, C. V.: Pathogenic organisms of the genus Listerella. Jour. Exp. Med., lxii (1935), pp. 203-212.

[11]Cromwell, H. W., Sweebe, E. E., and Camp, T. C.: Bacteria of the Listerella group isolated from foxes. Sci., lxxxix (1939), p. 293.

[12]Graham, R., Hester, H. R., and Levine, N. D.: Listerella from a premature bovine fetus. Sci., xc (1939), pp. 336-337.

The Swiss rank first as drinkers of milk, averaging 232 quarts per capita annually.

Trichinosis

Though considerable publicity has been given to the prevalence of trichinosis in man and swine in recent years, the disease in swine is much less prevalent than it was 35 years ago. As a matter of fact, the incidence is negligible in the mill-run of hogs, and clinical cases in man are rare.

Among the health problems confronting the veterinary service, trichinosis is by no means Public Enemy No. 1, as certain scareheads in the newspapers would lead the public to believe. It is just one among other potentials which the veterinary service is delegated to watch.

Strongylus Vulgaris Incidence

In a series of observations upon 174 horses killed at a meat-packing establishment in midwestern North America, 84 per cent were found to be infected with *Strongylus vulgaris* in the cecum. The majority of these subjects were aged horses, which confirms previous observations that there is a lack of age resistance to this parasite. (*J. H. Whitlock. Studies on Strongylus Vulgaris. I. Incidence of S. Vulgaris in Mid-Continental North America and the Reaction of the Infested Cecu. American Journal of Hygiene, section D, ..xxix, 1939, pp. 83-87.*)

Horses in the Invasion of Poland

NEWSPAPERS, newsreels and communiqués extolled mechanical transportation for its efficiency when the German legions invaded Poland and took that country under the wing of the Reich, but nothing was said about the 250,000 horses used to aid in carrying out that military operation. Though we can not disclose the source of the information, the number of animals reported to have been used in that expeditionary force is unquestionably authentic.

The movement of horses from North and South America to trans-Atlantic countries at the present time, in addition to the report from Poland, should be proof that the armed forces of 1940 do not expect to be horseless.

Pneumonia in the Thoroughbred*

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ON THE MODERN Thoroughbred farm pneumonia is considered a secondary complication, the result of a secondary invader, the primary cause being some filtrable virus affecting the upper air passages. Clinical evidence tends to bear out this belief, but there is an occasional case that may leave some doubt that pneumonia is always a secondary complication.

ETIOLOGY

Many eminent bacteriologists have concerned themselves with the study of pneumonia. Most authorities agree that the etiological factor in pneumonia is primarily a virus. Bacteriological examination of the nasal secretion and culturing of the exudate of the lungs disclose diplococci, streptococci, staphylococci, *B. coli* and numerous other secondary invading organisms.

There is one type of secondary pneumonia most often met with in practice among Thoroughbreds. It is adequately described as equine contagious pneumonia. (Equine contagious pneumonia is not to be confused with the much discussed contagious pleuropneumonia of the horse.) Equine contagious pneumonia is an acute inflammatory disease of the lungs, caused by a virus and later complicated by secondary invaders. It is characterized by lesions in the bronchi and the smaller bronchioles in its initial stages and, later, by independent areas of abscess formation and necrosis in the parenchyma of the lung, caused by the secondary invader, *Streptococcus pyogenes.*

Many workers classify equine contagious pneumonia as influenza, while others believe that it is an entity distinct from influenza. From a purely systematic viewpoint the following premises may be advanced:

1) Horses showing so-called pure symptoms of equine contagious pneumonia can transmit the disease to other horses, which thereupon show symptoms typical of influenza.

2) Horses which have been afflicted with influenza and which have subsequently recovered are immune to equine contagious pneumonia thereafter.

3) Horses suffering with equine contagious pneumonia do not seem to respond to the salversan treatment, whereas horses suffering with equine influenza, so-called, respond readily.

These statements seem merely to emphasize the fact that it is impossible to draw any strict and distinct delineation between equine contagious pneumonia and equine influenza.

Shütz of Germany, working with equine contagious pneumonia in the latter part of the 19th century, concluded that *S. pyogenes* is the cause of the disease. He produced the disease by injecting into the lungs of experimental subjects pure cultures of streptococci taken post mortem from lung lesions of affected horses. Although he failed to demonstrate the contagious nature of the disease, his work was accepted as authoritative for many years.

Some years later, attempts to produce an immunity in German army horses by the injection of vaccine made from *S. pyogenes,* or by the injection of specific immune serum, failed. New research was stimulated and plans were instituted to search beyond the streptococcus for an etiological agent. After many years of research, workers proved the disease to be of virus origin and revealed that the streptococcus organism was an agent of secondary effect. Further studies with the virus disclosed that this agent is only pathogenic for the horse and ass and that it is not very virulent outside the animal body. Attempts to produce the disease by intravenous and subcutaneous injections proved fruitless, but the disease could be produced by applying the exudate, taken from an infected lung

*Presented before the Section on General Practice at the 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1939.

lesion, to the mucous membrane of the nasal passages or to the mouth of experimental horses.

PATHOGENESIS

The incubation period of equine contagious pneumonia is believed to vary from 15 to 40 days. Successful attempts to transmit the disease seem to indicate that the above range may be accepted as tenable.

The disease manifests itself by a sudden rise in temperature, as high as 105 or 106° F. This temperature persists, generally, for about four days, after which, excluding possible secondary complications, it subsides.

Experimental demonstrations have proved that the virus may make its entry *via* the respiratory and digestive systems. Hematogenous and lymphogenous pathways of affection also have been alluded to the disease. The disease has never been definitely transmitted *via* the hematogenic or lymphogenic route, but it has been produced by the experimental application of diseased lung exudate to the mucous membrane of the nasal passages and the mouth of experimental subjects, manifesting itself in 15 to 40 days.

The wide range in the incubation period may account for the irregular appearance of the malady. Individuals at the extreme ends of a barn may simultaneously show symptoms, or again, individuals lined up in one, two, three order may manifest symptoms in a consecutive fashion. Other sundry and less important factors, such as incessant exchange of animals in a race-track barn or the conglomerate mixing of horses during shipment, also may be associated with the sporadic occurrence. It may be well to note that an animal which once recovers from the disease becomes heir to a rather permanent immunity.

The streptococcus is an ubiquitous organism in the equine species. It exists in the horse as a saprophyte or non-pathogen until suitable surroundings encourage its pathogenicity. The organism seems to have a peculiar property of attacking the mucous membrane of any part, especially when the mucous membranes are inflamed or congested. Streptococci have been isolated any number of times at autopsy from the bronchial passages of normal lungs of horses. Therefore, one can readily understand why the *S. pyogenes* is the most constant offender in the induction of secondary complications in so-called virus pneumonia. The virus follows the respiratory route, gaining entrance through the nasal passages or the mouth. It matters little which portal of entry it chooses, as long as it gains access to the trachea and more deeply situated respiratory structures.

The mucous membranes are susceptible to attack and the congested condition of portions of the lung offers an ideal medium for the rapid propagation of the streptococcus. The organisms attack the parenchyma of the lung and the subject begins to discharge a rust-colored, then a yellowish, cheesy exudate from the nostrils. Numerous small, isolated, independent, necrotic foci in the lungs grow larger and coalesce to form one large mass of diseased tissue. For all clinical purposes the patient then assumes a lobar rather than a lobular type of pneumonia and is subject to all sundry complications of this type of infection.

SYMPTOMS

If the patient has been carrying a temperature, and pneumonia is a secondary complication, there is a further rise in temperature. The fever is usually continuous, remaining up for seven to nine days. Cough, if present, is short and painful and dry at first, but it becomes moist and loose as the stage of resolution is reached. Nasal discharge may or may not be present, but in the stage of red hepatization, or second stage, there is sometimes a rust-red or prune-juice colored discharge which later becomes yellowish.

At first the pulse is not affected as much as the high temperature might lead us to expect; but as the disease progresses, due to the inflammation of the heart muscles, the pulse rate increases rapidly and the nostrils become distended. In severe cases the mucous membranes are highly congested, and in fatal cases they have a mahogany color, due to absorption and accumulation of toxins and lack of sufficient oxygen in the blood stream. Icterus is seen frequently

as the result of simultaneous gastrointestinal catarrh or to hemoglobinuria from a septic infection.

In the first stage ausculation determines the presence of rales on inspiration, which later pass away as that portion of the lung tissue becomes filled with exudate. Tubular and bronchial breathing are noted. Rales are noted again in the later stages, when resolution is taking place and the exudate is being expelled or absorbed. Percussion and ausculation vary with the stage present. In the earliest stages of congestion there is little that can be detected by percussion. This is true especially if the pneumonia involves only the central portion of the lung.

There is always some stomatitis. In hot weather the animal stands over the water container and thrusts the muzzle well under the water. Urine is scanty and highly colored, is very rich in urate and usually does not contain sugar, but during the febrile stage there is some albumen, which usually disappears when the temperature returns to normal.

Bowel action is retarded; constipation may be present and the feces coated with mucous. When there is much involvement of the nervous system as a complication, the rectum remains open due to the relaxation of the sphincter muscles. A severe infection with a high, persisting fever exhibits, as a result, an intense degeneration of the cardiac muscles; then cardiac symptoms are pronounced, the pulse being rapid and weak and often very irregular. Cyanosis of the nasal, buccal and lingual membranes is observed. When these cardiac symptoms and lesions exist, the pathological symptoms are more rapid and more pronounced, and the disease terminates fatally.

COMPLICATIONS

Pleurisy is a common complication leading to effusions in the chest with displacement of the heart, pain and characteristic dyspnea. The adhesions present may cause permanent injury or lead to death. Abscess and gangrene of the lung are to be looked for also, with their usual symptoms, nephritis and jaundice. Cerebral and meningeal complications as well as tendo-vaginitis and laminitis may be complications. Purpura hemorrhagica may occur during convalescence.

PROGNOSIS

The prognosis must always be guarded. The behavior of the heart during the attack is of greatest importance. A continued high pulse rate is of grave consequence. The extent of the involvement of the lung tissue is important. Early toxicity and involvement of the respiratory centers of the brain and cord are nearly always fatal. It is absolutely necessary that the heart be watched carefully and its normal action sustained as nearly as possible.

TREATMENT

Of first and prime importance in the treatment of pneumonia is the comfort of the patient. Clean, well-ventilated stalls free from flies, insects and decaying animal matter are a necessity. Next in importance is an intelligent, diligent groom to give careful nursing without exciting or worrying the patient.

The severity of an attack may be intensified by exposure, fatigue and unsanitary quarters. Horses which are worked or exercised after the beginning of an attack or before complete recovery are very likely to die. They should be placed alone. Avoid any disturbance and take every precaution to avoid any kind of exertion on the part of the patient. The careless administration of medicine in a drench and the unskilled use of a dose syringe contribute to the death toll in this disease.

The skill of the veterinarian is taxed greatly in the treatment of a severe case of pneumonia. The condition of the patient may change rapidly. This calls for careful observation so that serious complications can be foreseen and handled properly. Many authorities make the statement that drugs are useless and in many cases absolutely harmful in the treatment of lobar or lobular pneumonia. With such statements I heartily disagree. In my opinion, many drugs are a decided aid to the veterinarian.

The heart action and tone are a barom-

eter of the physical condition of the patient and should be carefully checked at least twice daily. Often a single dose of some form of digitalis will suffice to correct the tone, force and frequence of the heart action. In the initial stage of pneumonia such drugs as alcohol, adrenalin and pituitary extract, which act as vasoconstrictors, are indicated. Sodium bicarbonate and alkalis tend to reduce acidity and overcome toxicity. Blood transfusions and saline infusions tend to prevent exudation of blood serums in the congested lung. Shock, characterized by circulatory failure with accompanying suppression of body functions due to intoxication and infection, that may prove fatal must be looked for in the congestive stage.

Due to the fact that lobar pneumonia is due primarily to a virus, blood transfusions and hyperimmune and convalescent serums are indicated. Sulfanilamide and neoprontosil, which have given good results in cases of streptococcic infections, are certainly due consideration over some of the older and time-tried preparations; but, for me, they have not given superior end results.

Neoarsphenamine, a near specific for influenza cases in the initial or virus stage with or without very limited pulmonary involvement, does not give the results expected or desired, and I am of the opinion that this preparation is absolutely contraindicated in any case showing pulmonary lesions.

Sulfapyridine, a compound having a structural formula somewhat similar to sulfanilamide, has given excellent results in certain types of pneumococcic pneumonia in the human, but it does not seem to have the selective bactericidal action upon streptococci that it has upon pneumococci. Consequently, the results from its use in the treatment of pneumonia in the horse have not been comparable with those secured in the human. However, it must be said, in justice to sulfapyridine, that the beneficial results in some few cases of bronchial pneumonia in the foal have been amazing.

Oxygen administered through an improvised oxygen muzzle is practical and beneficial in the later stages of pneumonia and should be encouraged whenever the economic value of the patient justifies its use. The same can be said of CO_2 inhalations.

In the later stages of pneumonia, drugs that act as vasodilators are indicated. Resorbents, such as potassium iodide or some form of the soluble iodides, are indicated. Sodium chloride, sodium bicarbonate, sodium sulfate and ammonium chloride help to dissolve and liquefy the catarrhal secretions and croupous exudate in the lung, increase metabolism, thus increasing resorption, stimulate the secretory and excretory activity of the glands, and at the same time neutralize and dissolve acids which have collected in the animal body.

Lyophile Process of Preserving Biological Products

The preservation of biological products in desiccated form—by rapid freezing at a very low temperature, then dehydrating from the frozen state by means of a vacuum process, and sealing perfectly against the admission of air and moisture—is believed by high authorities in the field of biological production to be the coming method of marketing the antiserums, normal and convalescent serums, viruses, enzymes and toxins now so widely used in the practice of human and veterinary medicine.

"Lyophile" (solvent-loving) is the name applied to this new class of biological products. Desiccation *in vacuo* from the frozen state is said to reduce the cost of these products, and in veterinary medicine it will complicate their use to such an extent that laymen untrained will be apt to avoid them.

The strain of typhoid bacillus (*Eberthella typhi*) used for the production of typhoid vaccine was isolated from a fatal case in 1905 during the Boer War. The strain was obtained from Wright of England by Rawlings of Illinois. [*It is interesting to recall that Rawlings was the first professor of bacteriology at the McKillip Veterinary College.*]

Immunological and Histological Studies on Mink Distemper *

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IN PAST YEARS, epizoötics of a distemper-like disease have occurred from time to time on mink ranches in various sections of the United States. During the spring of 1938, ranches in New England and in the State of New York suffered from unusually severe outbreaks of this disease, losses in several instances amounting to 50 or 60 per cent of the entire herd. Somewhat later in the year, the disease appeared on a number of ranches in the western states.

No careful study of this disease appears to have been made. Attempted control measures have consisted of the administration of commercial canine distemper-immune serums and tissue vaccines. The use of these commercial biological products, made from the blood or tissues of dogs, has not appeared to influence the course of the epizoötics in any instance. This result might have been predicted from a consideration of two facts brought out by previous work on distemper.

First, Laidlaw and Dunkin,[1] in their studies which are now classical, demonstrated that tissue vaccine prepared from dog spleens, proven capable of immunizing dogs against canine distemper, was of practically no value in protecting ferrets against canine distemper. These workers also showed that canine distemper tissue vaccine prepared from ferret tissues was not effective in immunizing dogs against canine distemper. They found, moreover, that immune serum, prepared from the blood of hyperimmunized dogs, and definitely effective for dogs, was of no value when used in ferrets.

Second, Slanetz and Smetana,[2] studying a strain of distemper in ferrets, found it to be immunologically distinct from canine distemper. This suggests the importance of using a vaccine made from the identical strain of virus against which protection is desired.

Because of the urgency of the practical problem of controlling the disease, it was decided to test the effect of tissue vaccine on a number of ranches suffering from epizoötics in various stages of evolution and, for the reasons given above, to prepare the vaccine for each ranch from the tissues of animals dying on that ranch. In this way it was hoped that possible inefficiency resulting from both of the above considerations would be obviated simultaneously.

The clinical results of the use of vaccine prepared in this manner and the results of a few controlled laboratory experiments are presented below.

Clinical Picture and Course of the Disease in Minks

Adult Animals.—The typical picture in the adult mink was similar to that of canine distemper in the ferret. Refusal of food for several days, with a watery or mucoid exudate on the conjunctivae, was usually followed in a few days or weeks by marked swelling and incrustation of the footpads and by edema, redness, vesicles, and incrustation of the lips and nose. In the later stages, loss of hair, progressive emaciation and permanent gluing together of the upper and lower eyelids were prominent signs. About 25 per cent of the adults showing these symptoms eventually died, usually between the tenth and 21st days after the onset of symptoms.

Animals in which the swelling of the footpads appeared first seemed to recover more frequently than those in which conjunctival exudation was the initial sign. Abortion was the rule in affected pregnant females, and necrotic fetuses of various sizes, from a few millimeters up to full-term size, were found upon postmortem examination in a number of animals. In those instances where live young were produced,

*From the St. Louis University school of medicine. This work was done in the department of pathology at the Harvard medical school, with the aid of a grant from the American National Fox and Fur Breeders' Association.

it was of interest to note that the gestation period (calculated in most instances from the date of a single coitus) was prolonged about 25 per cent above the normal, although the young were not above the average in size or development.

A number of adults (about twelve in all) recovered from the acute phase of the disease and lived for several months in an emaciated state. These animals suffered from spasmodic contractions of the abdominal and other groups of muscles, and in some cases from muscular incoördination and constant athetoid movements. Several of these animals died spontaneously after suffering from these symptoms for two or three months, and the remainder were killed. No inclusion bodies were found in the tissues of any of these animals and attempts to recover the distemper virus from the brain or other organs by injection into ferrets were not successful. In two instances, however, focal collections of neuroglia cells and areas of demyelinization of the white matter were found in the cortex and basal ganglia.

Kits.—In the young minks, or kits, the clinical picture was essentially like that in the adults, but with more severe symptoms, as a rule. A number of kits died suddenly without having shown any symptoms of the disease, except failure to eat for a few days. That death in these instances was due to overwhelming distemper infection is indicated by the fact that extremely numerous inclusion bodies were found in nearly all of the organs in such animals.

The mortality in nonvaccinated kits showing signs of the infection was 100 per cent, and no kits survived long enough to develop the neurological symptoms described above.

TRANSMISSION OF THE DISEASE TO FERRETS

In the initial experiment, eight normal ferrets, 5 months of age, were obtained, four of which were isolated in each of two previously unoccupied rooms. After an observation period of three weeks, during which all of these animals remained well, one group was injected subcutaneously with emulsified lung tissue from an infected mink (1 cc. of a 1:10 emulsion). All four injected ferrets died on or before the 21st day, while the controls remained well. The symptoms and signs in the injected ferrets were like those described above in minks, except for the constant occurrence in the ferrets of a copious mucopurulent nasal discharge and of extensive hemorrhage from the mouth and nose just before death.

Further observations on 38 ferrets inoculated with mink distemper indicated that the susceptibility to and the mortality from the disease in ferrets are both 100 per cent in unvaccinated animals, when injected with large doses of virus.

ATTEMPTS TO DETERMINE THE NATURE OF THE DISEASE

Histological examination of the tissues of more than 100 minks dying from or killed during the course of the disease showed the characteristic intranuclear and intracytoplasmic inclusion bodies of distemper. This cytological picture has been adequately described in dogs by De Monbreun[3] and in ferrets by Slanetz and Smetana.[2] The inclusions seen constantly in injected mink tissues were identical in appearance with those described by the above workers and their distribution in the various organs corresponded accurately with that described in ferrets by the latter workers.

The two strains of distemper which Slanetz and Smetana studied in the ferret, one of which was canine distemper, showed inclusion bodies identical in appearance and distribution, in spite of an apparently complete absence of cross immunization. The microscopic diagnosis of distemper can be made with great precision, but strain differences, according to our present knowledge, are not distinguishable by this method and can be determined only by immunological experiments.

There are many reasons for believing that the distemper virus is highly unstable and that modifications in its pathogenic and immunological properties may occur rapidly with residence in different species of animals. Reference already has been made

to the observations of Laidlaw and Dunkin bearing on this point. R. G. Green of the University of Minnesota, in a personal communication, stated that distemper virus from the fox, when passed serially through ferrets, soon becomes incapable of producing severe infection in the fox, and can then be used successfully for the immunization of foxes, just as modified small pox virus from the cow has been used to protect human beings against this disease. It seems possible that the control of distemper is to be sought along these lines, the only danger of this method lying in the possibility that the virus may at times resume its full virulence for the fox.

Two puppies, approximately 3 months of age, after a negative observation period of one week, were inoculated subcutaneously with 2 cc. (0.5 dr.) each of a 10 per cent lung suspension from an infected mink. Ten days later, both animals developed typical symptoms of distemper, and both recovered after a prolonged illness. These animals were later proved immune to large doses of canine distemper virus.

Another group of five puppies died of fulminating distemper during the observation period, obviously having been infected when purchased.

It is clear that no definite conclusions can be drawn from these experiments, but it seems likely that the first two animals may have been successfully infected with mink distemper, and if so, their subsequent immunity to canine distemper would indicate a close relationship between these two strains.

Two healthy adult minks from ranches on which no distemper had occurred for several years did not react in any way to large doses of commercial canine distemper virus.

Four ferrets were vaccinated against the strain of distemper described by Slanetz and Smetana with three injections of 4 cc. (1 dr.) each at weekly intervals. When subsequently injected with the virus of mink distemper, all four animals died with typical symptoms of the disease, without any shortening of the incubation period.

Slanetz and Smetana described clinical differences between canine distemper and their disease in ferrets. A longer incubation period (ten days) and swelling and redness about the mouth and scrotum were noted only in the case of canine distemper. In ferrets inoculated with mink distemper, the incubation period varied from seven to twelve days, and definite swelling, redness and vesicle formation were seen around the mouth in all cases.

On the whole, therefore, the mink distemper which we studied seems to be more closely related to canine distemper than to the ferret strain described by Slanetz and Smetana, although further work is needed to define accurately its relation to other strains of canine, fox, ferret and mink distemper. It seems probable that several different strains may be encountered in minks and that new strains may be developed at any time.

METHOD OF PREPARING THE VACCINE

Tissue vaccine was made from the tissues of minks dying spontaneously or killed with chloroform when it was obvious that recovery would not occur. Care was taken not to use tissues of animals dead for more than four hours.

The method used to prepare the vaccine involved no new principles and was essentially that used by Laidlaw and Dunkin and, more recently, by Slanetz and Smetana. Lung tissue was used most extensively, but at times the trachea and bladder were added to the lung tissue. Vaccine also was prepared separately from the livers and spleens of many animals, but this material was not used on a scale large enough to warrant a statement regarding its efficiency as compared with the vaccine made from lung tissue.

For a time each tissue used for vaccine was checked histologically for inclusion bodies and vaccine material was discarded unless inclusion bodies were numerous. Since only about 10 per cent of adult tissues and less than 1 per cent of the kit tissues failed to show numerous inclusions, however, tissues from a number of animals were frequently pooled in the later work, and microscopic examination was carried out on only an occasional animal.

Once the diagnosis of virulent distemper is established by the finding of numerous inclusions in the tissues of a number of animals on any given ranch, it seems unlikely that a poor batch of vaccine would be obtained by pooling the tissues of animals subsequently dying on that ranch. As pointed out later, however, on one of 16 ranches on which a positive diagnosis of distemper was made, a much less virulent form of the disease was present, with a prolonged clinical course and a lower death rate. In animals killed or dying spontaneously on this ranch, inclusions were frequently so rare that a prolonged search was necessary to demonstrate them. Such ranches offer a somewhat different problem as regards vaccination, which might be solved by using vaccine from a ranch with more virulent disease, or perhaps by preparing vaccine from ferret tissues.

The method of standardizing the vaccine by the number of inclusion bodies observed microscopically is much simpler than that of estimating the virus content by animal inoculation in various dilutions, and is believed to be sufficiently accurate for practical purposes. The more tedious method of titration reveals only the concentration of living virus, and it is possible that much of the virus present in living tissues already may be inactivated, but still capable of producing immunity.

In the actual preparation of the vaccine the tissue was removed from dead animals aseptically and finely ground to a thin paste in a sterile mortar with the addition of fine quartz sand and a small quantity of sterile physiological salt solution (0.9 per cent NaCl). This paste was then diluted with eight or nine volumes of the sterile salt solution and, after the sand was allowed to settle for a few minutes, filtered through several thicknesses of sterile gauze. Three-tenths per cent of full-strength (40 per cent) formaldehyde solution was then added. This suspension was subsequently transferred to sterile vaccine bottles which were stored in the ice box for four days before use. The bottles were shaken and inverted daily during this period. The vaccine was injected subcutaneously in the groin, two or three injections of 2 to 4 cc. (0.5 to 1 dr.) each, depending upon the size of the animal, being given at weekly intervals.

The lungs from an adult mink usually furnished enough vaccine for the injection of 30 to 50 minks, while the lungs from a kit were sufficient for injecting 15 to 25 minks. Since the tissues of kits practically always contained extremely numerous inclusions, it is probable that vaccine made from such tissues could have been used in greater dilution.

RESULTS OF VACCINATION OF MINKS

The method of vaccination outlined above was carried out on nine different ranches and, in all, more than 4,000 animals were vaccinated. Localized swelling at the site of injection of the vaccine occurred in less than 1 per cent of the injected animals and no deaths occurred which were attributable to the vaccination. After we had killed the sickest animals to make vaccine, all animals not obviously dying were vaccinated.

On some of these ranches, the epizoötic had been in progress for many weeks, while on others it was just beginning at the time that vaccination was begun. In most instances the process of vaccination required about two weeks. On each of the nine ranches, the number of new cases observed per day showed a sharp drop during the second week of the period of vaccination and, within a week after the completion of the process of vaccination, the epizoötic, from the standpoint of new cases, had come to an abrupt termination. The daily death rate also fell sharply at about this time but occasional deaths occurred for several weeks after this, representing, with very few exceptions, animals which showed symptoms at the time that vaccination was begun. This sudden termination of epizoötics in midsummer is contrary to past experience and is believed to be, in all probability, a result of the vaccination.

Statistical data from one ranch are given below. Although the course of the epizoötic following the vaccination was essentially the same on all nine ranches, this ranch is selected for a detailed report because data of particular interest were obtained on it.

Ranch C.—The animals on this ranch comprised 560 adults (413 females and 147 males) and 850 kits, the latter being born during the period of the epizoötic. (A few kits which died shortly after birth have been disregarded.) The first case was noted on February 10, 1938, but distemper was not suspected at that time. Between February 20 and April 28, when vaccination was begun, the death rate in the adult minks increased steadily. There were about two deaths per day at that time. Of the 850 kits born during this period, 25 died before vaccination was begun. Of the remaining 825 kits, all of which were vaccinated, 133 died. Twenty of these kits which died in spite of vaccination were apparently free from symptoms at the time of the first injection of vaccine, but 19 of them developed symptoms before the second injection. The remainder (113) were sick at the time of the first injection.

Only one kit which was free from symptoms at the time of the second injection (the final injection on this ranch) subsequently became sick. In this case, symptoms appeared one week after the second injection. This kit was one of a litter of ten which had not been separated from their sick mother. The other kits in this litter, vaccinated at the same time as the mother and the kit which died, showed no symptoms at any time. The mother recovered after a prolonged illness.

The total number of adults which died was 75, 50 dying before vaccination and 25 after vaccination. Every one of the 25 adults which died in spite of vaccination was sick at the time of the first injection. In other words, not a single adult acquired the disease after vaccination.

Of the above minks, one group of 60 adults and 114 kits were housed in out-of-door cages, about 200 yards from the sheds in which the remainder of the animals were kept. Foster kits from mothers dying in the shed were distributed among six litters in this group during April and May. These foster kits all died, but since no evidence of infection had been noticed in the 60 adults and 114 healthy kits, these animals were not vaccinated when those in the sheds were injected.

On June 30, six weeks after the complete termination of the epizoötic in the sheds, new cases of distemper began to appear in the unvaccinated animals in out-of-door cages. Cases were noted both in the cages in which foster kits had died and in cages where foster kits had not been placed. At this time all of these previously unvaccinated animals were vaccinated. Twenty kits and nine adults from this group, all of which were sick at the time of vaccination, died. No new cases were seen in this group after the second injection of vaccine was given.

Certain other statistics from this group of animals will be given because of their interest from the epizoötiological point of view.

Litter 1.—Five healthy kits and healthy mother. Two apparently healthy kits from a sick mother in the sheds were added to this litter on April 30. These two kits died three weeks later. The foster mother and her five kits died during the month of July, in spite of vaccination on June 30 and July 8, but all were definitely sick before the second vaccination.

Litter 2.—Two healthy kits and healthy mother. Two foster kits from a sick mother were added to the litter. All four kits and the foster mother eventually died. All were sick before vaccination.

Litter 3.—Two healthy kits and healthy mother. Two foster kits from a sick mother were added to the litter. The two foster kits died, while the mother and her two kits remained healthy, although they were not vaccinated until two weeks after the second foster kit died.

Litter 4.—One healthy kit and healthy mother. One foster kit from a sick mother was added to the litter. The foster kit died, while the mother and her kit remained well, although not vaccinated until 27 days after the foster kit died.

Litter 5.—Three healthy kits and healthy mother. Two exposed foster kits were added. Both foster kits died shortly after being placed in this litter and two of the three other kits died late in July. One kit and the mother showed no symptoms although not vaccinated until 30 days after the two foster kits died.

Litter 6.—Three healthy kits and healthy mother. Two exposed foster kits were added. These two foster kits died, but the mother and her three kits remained well, although they were not vaccinated until three weeks after the second of the foster kits died.

On all ranches, every nonvaccinated kit showing symptoms of any sort succumbed to the disease. On ranch C, two kits which showed definite symptoms at the time of the first vaccination eventually recovered. On another ranch, 19 kits showing symptoms at the time of the first vaccination made apparently complete recoveries. These observations are in agreement with those of Slanetz and Smetana, who found that vaccination of ferrets in the early stages of illness had some apparent curative effect. A large number of vaccinated adult minks, sick at the time of vaccination, made complete recoveries, but this fact is not significant because a certain number of adults recover from the disease without treatment. It is only in ferrets and in mink kits, where the natural mortality is 100 per cent, that recoveries following vaccination may be attributed to the vaccine.

LABORATORY STUDIES

A number of healthy minks from ranches free from distemper for several years were contributed for experimental study, and it was originally planned to carry out a controlled test of the vaccine used in the groups previously described. Preliminary tests, however, showed a low susceptibility of these minks to the virus of mink distemper. Only one of six minks injected with heavy doses of virus developed the disease and died, and this animal was found also to be suffering from coccidiosis, parasites being present not only in the intestine but also in the liver.

It was therefore decided that an accurate test of this method of vaccination for minks would require the use of mink kits, in which, as pointed out, the mortality is 100 per cent, and in which it seems probable that the susceptibility also might be 100 per cent should overwhelming doses of virus be used.

Since the susceptibility and mortality of the mink distemper in ferrets had been shown to be 100 per cent, it seemed that something could be learned by testing the ability of mink-distemper vaccine made from mink tissues to protect ferrets against mink distemper. For purposes of comparison, in addition to normal controls, a series of ferrets injected with large doses of commercial canine distemper tissue vaccine (made from the spleens of infected dogs) also were included in the series.

Ferrets were shipped to an isolation ranch in the country and held for observation for at least a month before vaccination was begun. All vaccinations were carried out at that ranch. Two injections of 3 cc. (0.1 oz.) each were given ten days apart in the case of the mink-distemper vaccine, and two injections of 5 cc. (.16 oz.) each in the case of the commercial canine distemper vaccine. Ten days after receiving the second injection, each group of vaccinated animals, together with an equal number of controls, were removed to a room at the Harvard medical school, where they were injected with the virus of mink distemper.

In all, 15 ferrets were injected with the mink-distemper vaccine (made from animals dying on the same ranch from which the mink-distemper virus originated), ten ferrets were injected with commercial canine distemper vaccine, and 22 controls were removed to the medical school and infected with mink distemper. In the case of one group of six control animals, no injection of live virus was given, but all of these acquired the disease by contact.

All 22 of the control ferrets and all ten of the ferrets vaccinated with commercial canine distemper vaccine became ill after an incubation period of seven to ten days, showed the typical clinical picture previously described, and died before the 22nd day after injection with the virus (or exposure to it, in the case of the group of six controls mentioned above).

The 15 ferrets injected with the mink-tissue vaccine did not begin to show symptoms until the 21st day after injection; in most groups, not until after all of the control animals died. The disease appeared distinctly milder in this group of ferrets, and seven of the 15 animals in this group recovered. Histological examination of the tissues of several of the animals dying in this group, however, showed inclusion bodies to be as numerous as in the other two groups.

As already stated, four ferrets vaccinated with tissue vaccine made from ferret tis-

sues infected with the Slanetz strain of ferret distemper were included in this series. These animals all reacted like the controls with typical infections terminating fatally before the 22nd day after injection.

BACTERIOLOGICAL STUDIES

Tissues from minks dying or killed with chloroform when moribund from distemper were studied bacteriologically, both on ordinary culture mediums and on the modified Maitland culture medium recently devised by Zinsser.[4] The surprising feature of this study was the infrequency with which secondary bacterial invaders were found, even when lung tissue was cultured. In only two out of ten animals were bacteria found —in both cases staphylococci. This is in striking contrast to the results of similar studies on the tissues of foxes suffering from distemper. From these fox tissues a variety of secondary invaders were cultured, including *Bacillus bronchisepticus* and an atypical organism of the paratyphoid group.

In agreement with these bacteriological studies is the fact that histological evidence of secondary infection was lacking in the tissues of more than 100 minks studied, while in each of the 28 foxes studied, suppurative pneumonia or focal abscesses in the liver were seen. From the data obtained, the conclusion must be drawn that minks commonly die from the virus of distemper alone, while secondary invaders are of primary importance in the fox, as they frequently are in the dog.

From the tissues of one mink, chopped in tyrodes solution and spread on Zinsser's agar-slant medium,[4] a minute spiral organism similar to the organism of bovine pleuropneumonia was cultured.

This organism was of interest for two reasons. First, it seemed identical with an organism described by Schoetensack[5] as the cause of distemper in dogs. Second, when grown in tissue cultures, this organism produced spherical colonies in the cytoplasm of mesenchymal cells, which had somewhat the general appearance of inclusion bodies, although they were never completely homogeneous.

Since Schoetensack has claimed to produce immunity to distemper by vaccination with his organism, six ferrets were injected with large amounts of blood-broth culture of the spiral organism. No symptoms were produced in these ferrets, and injection with mink-distemper virus five weeks later indicated that no immunity had been produced.

On one occasion this spiral organism was recovered from the brain of a mouse six days after intracerebral injection, but no true pathogenicity could be demonstrated. The organism is morphologically similar not only to the etiological agent of bovine pleuropneumonia and to the organism described by Schoetensack in distemper, but also to an organism recently found by Sabin[6] to cause arthritis in mice, and to several other organisms recently isolated from animal tissues under a wide variety of conditions. On the whole, it seems unlikely that an organism of this type could be etiologically related to distemper, as Schoetensack believes, but it is conceivable that the virus of distemper might be able to grow symbiotically with it under certain conditions. It should be mentioned that Schoetensack does not find virulence or immunizing properties associated with all strains of the spiral organism which he recovers from distemper material.

HISTOLOGICAL STUDIES

In the course of this work, a large amount of histological material has accumulated. It has been mentioned above that the only histological finding of importance in mink tissues was the presence of inclusion bodies, both in intracytoplasmic and intranuclear, which corresponded completely, both in appearance and distribution, with those described by Slanetz and Smetana. It seems unnecessary to describe these inclusions, since they have been adequately described and illustrated by various workers.

Inclusions were not found in the central nervous system, but in rare instances focal collections of neuroglial cells and areas of demyelinization were noted in the brain, suggesting that the virus may have been

present there at some previous time. This appearance was seen only in recovered minks exhibiting chorea-like symptoms.

In about 10 per cent of the minks examined, a specific type of focal pneumonitis was seen. Fecal areas of consolidation, often 2 to 3 mm. in diameter, were found to be due not to an accumulation of exudate in the alveolar spaces, as in ordinary pneumonia, but to marked proliferative activity on the part of the histiocytes in the alveolar walls. These cells were greatly enlarged, often multinucleated, and contained large numbers of typical inclusion bodies, both intracytoplasmic and intranuclear. No evidence of bacterial infection was found, and this lesion is undoubtedly caused by the virus of distemper alone. A similar lesion was described in the lungs of dogs by De Monbreun.[3]

The importance of finding inclusion bodies for the rapid and accurate diagnosis of distemper is to be emphasized, since it has apparently not been appreciated. The inclusions are perfectly characteristic and, with the exception of animals coming from one ranch, in which the disease was clinically mild, have always been present in large numbers. It should be mentioned also that inclusions were numerous in the tissues of the majority of the foxes examined but in several foxes typical inclusions were absent from the lungs, and found in the liver only after prolonged search.

DISCUSSION

The empirical results of the vaccination of minks against distemper with the ranch-made vaccine described furnish strong evidence of its efficiency in controlling the disease, and the results of the laboratory experiments detailed lend definite support to this view. Unfortunately, the experimental work was interrupted, and much still remains to be done.

In view of the definite partial protection afforded to ferrets with vaccine made from the virus of mink distemper in mink tissues, and in consideration of the relatively low susceptibility of the mink, it seems possible that an effective vaccine for minks might be prepared from ferret tissues. Best results, however, would be expected with a vaccine prepared from mink tissues, and such a vaccine could be tested accurately only by utilizing mink kits.

The alternative to the method described above would be the injection of living attenuated virus, prepared by the method recently used by Green in protecting foxes against distemper. The possibility of attenuating a strain of mink distemper, by passage through ferrets or other animals, to a point where it could be safely injected into minks, is worthy of consideration. Until such a method is worked out, it would seem that the method described in this paper offers the best chance of success. The apparently excellent results obtained are believed to be due in part to the relatively low susceptibility of the adult mink and to the apparent absence of complicating secondary infections, even in young minks.

SUMMARY

An epizoötic disease of minks, identified as a strain of distemper by histological studies, has been described clinically and shown to be probably more closely related to canine distemper than to the strain of ferret distemper described by Slanetz and Smetana. The probability that this disease differs somewhat from typical strains of canine distemper is, however, pointed out.

Two reasons for the inadequacy of commercial canine distemper antiserums and vaccine, prepared from the blood or tissues of dogs, in controlling epizoötics of distemper in minks are pointed out.

This strain of mink distemper was readily transmissible to the ferret and produced a uniformly fatal infection in that animal. The mortality in young minks without treatment was 100 per cent, and the susceptibility of young minks is probably high. The mortality in adult minks does not exceed 50 per cent, and the susceptibility of adult minks is relatively low.

A method of vaccinating minks with tissue vaccine, prepared for use on each ranch from animals dying on that ranch, is described. Apparently excellent empirical results from the use of this vaccine on nine mink ranches are reported.

Some evidence is introduced that the vaccine used had curative effects if given in the early stages of the disease to young minks, since at least 21 young minks survived under these conditions, as contrasted with a natural mortality of 100 per cent.

Minks apparently die from infection with distemper virus alone, in contrast to foxes, in which secondary bacterial infections play an important rôle.

Epizoötiological data, showing the variable results of placing exposed foster kits in healthy litters, are given.

Tissue vaccine prepared from mink tissue infected with mink distemper was partially effective in protecting ferrets against this same strain of mink distemper, causing marked prolongation of the incubation period, and a reduction of the mortality from 100 per cent to 50 per cent.

Commercial canine distemper tissue vaccine, prepared from the spleens of dogs suffering from canine distemper, was completely ineffective in protecting ferrets against mink distemper.

A minute spiral organism, similar to the etiological agent of bovine pleuropneumonia and also to the organism described by Shoetensack as the cause of distemper in dogs, was cultured from the tissues of one mink. This organism was capable of producing neither symptoms of nor immunity to mink distemper when injected into ferrets.

The importance of finding inclusion bodies, as a rapid and accurate method for diagnosing distemper, is stressed.

Practical considerations in the control of distemper in minks are discussed.

References

[1]Laidlaw, P. P., and Dunkin, G. W.: A report upon the causes and prevention of dog distemper. (Incorporated in a report issued by the American Distemper Committee, Ames Bldg., Boston, Mass.)

[2]Slanetz, C. A., and Smetana, H.: An epizoötic disease of ferrets caused by a filterable virus. Jour. Exp. Med., lxvi (1937), 6, pp. 653-666.

[3]De Monbreun, W. A.: The histo-pathology of natural and experimental canine distemper. Amer. Jour. Path., xiii (1937), pp. 187-212.

[4]Zinsser, H., Fitzpatrick, F., and Wei, H.: A study of Rickettsiae grown on agar tissue cultures. Jour. Exp. Med., lxix (1939), 2, pp. 170-190.

[5]Shoetensack, H. M.: Relation between canine distemper and *Asterococcus canis*, type I and type II. Kitasato Arch. Exp. Med., xiii (1936), pp. 269-280.

[6]Sabin, A. B.: Experimental proliferative arthritis in mice produced by filterable pleuro-pneumonia-like micro organisms. Sci., lxxxix (1939), 2306, p. 226.

The Speed of Animals

Although certain animals, such as the antelope, are reputed to "fly like the wind," their top speed seldom exceeds 35 miles per hour and this rate of travel can be maintained only for two or three miles. The antelope and the race horse (Thoroughbred) are the swiftest of animals. All others travel at a lower rate than these.

In wild life, speed is the best defense against the predatory enemies. But for its speed the rabbit could not have survived the hunger of the carnivorous fox, nor could the deer have protected itself against extermination by wolves. Though man can run a hundred yards in less than ten seconds, he can hardly make 15 miles an hour over a mile stretch. Animals are noted for having a quick "getaway." They hit full speed from the very start, whereas man must take several strides before striking his best momentum.

It is interesting to note the top speed of certain animals:

Animal	*m. p. h.*
Race horse	35
Antelope	32
Jack rabbit	30
Red fox	28
Greyhound	25
Deer	24
Coyote	21
Elk	16

A very small margin of speed is the factor that has preserved many species of animals through the centuries.

The Prairie States Forestry Project has laid out 10,800 miles of field wind breaks since 1935, and planted over 55 million trees last year.

The dog population of the United States is estimated to be approximately 12,000,000. About 3,000,000 are pedigreed and approximately 600,000 are registered.—*Dog World, February 1940.*

Black Widow Spider and Snake Venom in Small Animals

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Part I—The Black Widow Spider

LITTLE has been written on the subject of black widow spider venom in small animals. As I come in contact with a great deal of this malady in my practice, it occurs to me that many others have the same troubles and, perhaps in some cases, are unable to secure sufficient history or information to recognize the cause.

The black widow spider (=*Lactodectrus mactans*) is not by any means confined to my district in the California desert. It is said by authorities to be distributed practically over the entire United States as well as over other more southern climes.

As is the case with rattlesnakes, these spiders seem to prefer sparsely settled regions and old, deserted garages or homes. Other favorite spots are lumber piles or boards lying on the ground in dry areas. However, one should be a little careful in handling anything which has not been disturbed for some time, as they may be found in nearly any place which offers seclusion.

One clue as to the presence of this spider is her peculiar web. Most spiders build a web which is a beautiful thing to behold. However, true to her other characteristics, the deadly black widow builds a web as unbeauteous as herself. It is a conglomeration of crisscrossing strands about 8 to 12 in. wide and nearly as deep (depending on location), with neither rhyme nor reason apparent in its construction.

DIAGNOSIS

The after effects of a bite from this spider vary a great deal and unless a history is definite or nearly everything else can be eliminated, it is sometimes quite difficult to make a diagnosis. As a guide to diagnosis, therefore, these cases can be divided into four groups, as follows:

Peracute Type.—The peracute case usually is recognized quite easily. The bite, which causes extreme pain, produces violent emesis within an hour, the belly muscles become rigid, and there is swelling and general edema of jelly-like consistency around the area of the bite. The bite of the black widow differs from that of a rattlesnake in that it is difficult to find the fang punctures. Symptoms of pain are intense and in many cases necessitate complete anesthesia with nembutal to produce any relief for the patient. Symptoms of collapse are certain to manifest themselves within two hours; death may occur in four to six hours. This type is fairly rare.

Acute Type.—The acute type is similar to the peracute except that the pain is not quite as intense at first. The main symptom is soreness of the skin. This is so outstanding that it is quite easily recognized. The entire integument seems to be somewhat thickened and even slight pressure produces violent pain which causes the animal to scream or squeal for several minutes. There is general weakness and dyspnea to some extent, with a pinched, anxious expression about the animal. It resents being moved or touched. These symptoms may exist for 24 hours without apparent change, after which symptoms of gradual paralysis occur. These usually affect the legs first; however, any part or parts of the body may become affected. Death occurs within 48 to 72 hours.

Subacute Type.—The subacute type is sometimes difficult to detect. The first symptom of pain at the site of the bite is present, but this is not as intensified as it is in peracute cases. There may or may not be vomiting. The belly muscles are rigid, but not as markedly as in the peracute type. After about 36 hours, during which practically no outstanding symptoms can be observed, a severe arthritic irritation seems to occur. I have seen dogs come in limping on one leg which was sore in every joint; within five minutes they were limping on the other leg; and perhaps in five

minutes more, on still another leg. On manual manipulation all legs and joints of the legs seem to be very sensitive to pressure or any manipulations. The dog also may exhibit pain on being touched or picked up. The appetite may be impaired or completely lacking.

Chronic Type.—Type 4, or the chronic type, may occur in dogs following the acute or subacute types, or it may not even be noticed by the owner until the paralytic state is reached. Our usual history is as follows: The dog did not eat for a day or two and seemed a little sore, and would not jump up on chairs or the sofa. It slept a great deal but would move around if coaxed to do so. After a day or two it started to eat but became weak in either the back or front legs. It seemed a little wobbly at first and soon became unable to use the legs at all.

The animal usually appears to be in fair condition and has a good appetite, but it can not walk. Later, loss of control over defecation and micturition occurs. This paralysis may become progressive and finally cause death through uremic poisoning or autointoxication, rather than from any particular effects of venom that are noticeable on autopsy.

One important item unmentioned up to now is that a percentage of dogs seem to be immune to the bite. In experiments carried on last summer, we were unable to produce symptoms of any kind in many dogs which we permitted spiders to bite. We are not certain whether this immunity is natural or whether it is acquired through the medium of a former bite from which recovery resulted. This is true of some snake bites, as will be pointed out presently.

TREATMENT

We first used snake antivenin with practically no success and, therefore, resorted to other antidotes. Fatalities are great in the peracute type and we have found no effective treatment for such cases. Death occurs before treatment can take effect.

In the acute cases treatment is effective if instituted early. Ten to 50 cc. (.33 to 1.66 oz.) of blood from an immune donor is injected intramuscularly at several sites. This should be repeated in two to six hours, depending upon the progressive symptoms of the case.

Subacute cases respond most readily to treatment. The same treatment that is employed in the acute type is used here, but the interval between injections may be extended, if the symptoms warrant such procedure. In cases where collapse or extreme pain is not in evidence, the serum alone may be used intravenously, with good success.

In peracute or acute states, when the donor is available immediately, direct or indirect blood transfusion may be employed. First, however, the intense pain must be relieved. This is accomplished by administering nembutal. As an adjunct, 1/150 to 1/75 gr. of atropine sulfate may be given either simultaneously or beforehand as a heart stimulant principally to counteract shock to the circulatory apparatus brought about by nembutal plus venom.

At an early date we hope to have more definite data on this subject. At present, we are experimenting on a serum to be used in both human and animal cases. Our findings will be available in the JOURNAL as soon as we can construct and prove them.

RECOMMENDATIONS

1) It is well to be on the outlook for black widow spider bites, regardless of your location.

2) If you have a case or know of a case that has been bitten and recovered, keep it in mind as a possible donor.

3) If no known-immune donor is available, try serum from a large, well dog, not from a puppy. There is the chance that the former may be immune.

4) It is always better to give an unfavorable prognosis until at least four days have passed, since dog serum is not as yet a proved specific.

5) Cases of so-called posterior paralysis, without known cause, should be regarded as suspicious until spider bite can be eliminated as the causative factor.

6) Do not use morphine to relieve pain.

Part II—Rattlesnakes

Rattlesnake bites are a veterinary problem in the California desert. The most troublesome snake is the deadly sidewinder, or horned rattlesnake (=*Crotalis cerostes*). It seldom measures over 18 in. in length and has a color corresponding nearly to that of sand. Its rattle is very dim and not always used. Not being able to stand much sunlight, it travels only at night. I have seen bites from these snakes cause death to dogs of the smaller breeds within 45 minutes.

In most cases the bite produces a large, rather firm, doughy, edematous lesion. If the bite is located on a leg, in some instances it may be mistaken at first for a fracture, until careful examination reveals no broken bones.

This type of bite is painful, but becomes pronounced only on pressure; the fingers leave a distinct imprint lasting for several minutes. If the bite is located on the nose, as it frequently is, the face becomes unrecognizable in a few minutes. The bite of a horned rattlesnake is serious, as edema of the glottis is apt to follow, which causes a quick but horrible death.

All rattlesnake bites are dangerous and should be considered as such. At least, this is true of our western snakes. In some cases, apparent recovery may change to sudden cardiac and circulatory collapse after all other symptoms are eliminated.

TREATMENT

The general rule for treatment is immediate injection intravenously of antivenin (Mulford), either the concentrated or regular.

We usually start with a dose of 15 to 30 cc. (.05 to 1 oz.), depending upon the size of the dog. The location of the bite also is an important factor governing the dosage. Thirty cc. appears to be a minimum dose for a Scottish Terrier bitten on the nose or the face. The same dog would probably receive 15 cc. if bitten on the leg or body and would be watched carefully for signs of diffusion and disappearance of the edema. If we see no definite result or an increase in swelling within two hours, we repeat the dose or increase it. If the swelling subsides, it is well to give a second dose within twelve hours, since symptoms of cardiac disturbance or collapse sometimes appear after twelve hours have passed.

To those cases which have been bitten several times or are slow to respond we give two or three additional doses two days following the initial treatment.

One bite seems to confer a temporary immunity. At least, subsequent bites are of much less consequence than the first one.

RECOMMENDATIONS

1) Never underrate the potential seriousness of a bite.

2) Administer antivenin as quickly as possible.

3) Many grave cases will recover completely in four to six hours.

4) Watch for secondary symptoms following apparent recovery.

5) Avoid so-called antidotes, such as potassium permanganate, gunpowder and whiskey. Most of these have been proved to be detrimental rather than helpful.

Sodium Benzoate in Bronchopneumonia*

Sodium benzoate intravenously is recommended in the treatment of bronchopneumonia of dogs. It has antiseptic and antiputrid action in liberating benzoic acid and modifying the parenchyma by eliminating it *via* the respiratory tract. Moreover, sodium benzoate acts as a dializer, increases respiratory combustion, and facilitates the expulsion of toxic products. The treatment is contraindicated where there is renal congestion.

The dose is 4 Gm. (1 dr.) in 10 cc. (.33 oz.) of water, daily or twice daily.

The oxygen requirement of a fertilized mammalian ovum is 0.00,073 cmm. per hour.

A 10-watt electric light bulb requires a billion billion electrons per second to keep it lit.—*Science and Discovery.*

*Paul Maury. Thèse, Toulouse. Abst., Revue de Médecine Vétérinaire, xci, April 1939, p. 222.

Effects of the Intravenous Injection of Certain Salts of Sodium, Calcium and Potassium on Intestinal Tonus and Motility in the Dog*

By L. M. JONES, A.B., D.V.M., M.S., and E. A. HEWITT, B.S., D.V.M., M.S., Ph.D.
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FOR MANY YEARS saline purges have been administered by the mouth but, recently, certain salts also have been injected intravenously to stimulate the intestinal tract.

This study was undertaken with the object of determining the ability of certain sodium, potassium and calcium salts to stimulate the muscular activity of the intestine in the dog.

Two types of experimental procedure were followed. Type 1 required an anesthetized dog and involved a recording of the respiratory movements, of the blood pressure from the carotid artery and of the intestinal movements as revealed by a balloon placed within the small intestine. Type 2 provided for a recording of respiratory movements and a recording of intestinal movements as revealed by a jacket placed about an exteriorized skin-covered loop of small intestine.

EXPERIMENTAL PROCEDURE

In type 1, the dogs were anesthetized with pentobarbital sodium dissolved in distilled water and administered by intrapleural injection at the rate of 30 mg. per kilogram (2.2 lbs.) of body weight. A 6 per cent solution was used.

The records were made on long smoked paper revolving on a Miller kymograph. Blood pressure was recorded by a mercury manometer connected by means of a three-way cannula with the left carotid artery. The cannula also was connected with a pressure bottle containing an anticoagulant (8 per cent sodium citrate solution).

Respirations were recorded by means of a pneumograph connected with a Becker air tambour. Movements of the intestine were recorded by the balloon method. By means of a median incision the anterior portion of the jejunum was isolated and exposed. A thin rubber balloon attached to a human catheter was inserted into the lumen through a small incision in the intestinal wall. The incision in the wall of the intestine was closed by a purse-string suture which was drawn tightly around the catheter connected to the balloon. The intestine was replaced in the abdominal cavity and the abdominal incision was closed by clamping with hemostats. The catheter was attached by tubing to a burette partly filled with water; the water was allowed to flow into the balloon, thus distending it. The top of the burette was connected by rubber tubing with a Macey air tambour. The movements of the intestine exerted a pressure on the water in the balloon that was transmitted to the column of water in the burette. The column of water compressed the air in the top of the burette and transmitted the variation to the recording tambour.

A burette for injection purposes was connected by means of rubber tubing to a three-way cannula inserted into the femoral vein. A three-way cannula was used so that it could be washed out between injections, if necessary. Special pipettes to control the rate of injections were inserted in the tubing connecting the burette with the cannula. The pipette most frequently used allowed 100 cc. (3.3 oz.) of the solution to pass in eight minutes.

In type 2, a method that is new in the study of intestinal motility is based on an operation on the dog first performed by Biebl[1] and improved by Bors and Polano.[2] The method used in these experiments was adapted largely from the technic of Bors

*From the department of veterinary physiology and pharmacology, Iowa State College. Presented before the Section on Research at the 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1939.

and Polano. Two parallel skin incisions, about 6 in. in length, were made longitudinally about 1½ to 2 in. apart in the loose skin of the right flank. The subcutaneous fasciae were torn loose underneath the strip of skin on all sides. A 3- or 4-in. incision through the abdominal muscles and peritoneum was made underneath the median skin wound. The first part of the jejunum was carefully located and brought through the opening in the abdominal wall. A section of mesentery was selected that was comparatively free of blood vessels and nerves. The section was cut away from the intestine and care was taken not to injure neighboring vessels or nerves. The strip of skin was folded around the free loop of intestine and the edges fastened together with interrupted silk sutures. The peritoneum above was sutured with catgut; then the abdominal muscles in like manner. The free skin lateral to each of the parallel incisions was stretched until both parts met underneath the loop and were sutured with silk.

The wounds were protected by a double layer of sterile gauze cut in two sections to fit the area and held in place by special adhesive cement. The bandage was examined on the second day following the operation and loosened if it was too tight; it was removed on the third or fourth day. The silk sutures usually were removed a few days later. A broad fiber-board collar was used to keep the dog from annoying the wound.

In 1½ to two weeks following the operation the wound was sufficiently healed so that it could be measured and a metal jacket was made to fit the intestinal loop of the individual dog. The jacket was made of a hollow, metal cylinder that was cut down the middle from top to bottom and hinged so that it would open to permit the entrance of the intestinal loop. The cylinder was about one-fourth inch larger on all sides than the skin-covered intestine. At both ends of the cylinder a metal collar one-fourth inch wide was soldered so as to fit moderately close about the intestine and to close the end of the cylinder. A metal tube about one inch in length and one-half inch in diameter was soldered to an opening at the middle of the cylinder. A rubber balloon was attached to a three-eighth inch rubber tubing by a rubber band and the balloon was cemented inside the metal jacket. The rubber tubing was put through the short metal tube leading from the side of the jacket and attached to a water manometer with a writing point. The rubber balloon and tubing were carefully emptied of all air and filled with water. A water pressure of about 15 cc. (0.5 oz.) was exerted on the balloon.

While the wound was healing, each dog was trained to lie quietly on the table. The dogs maintained their positions for periods sometimes as long as 1½ hours without appreciable movement. Some of them permitted repeated intravenous injections in the cephalic vein without interfering with the recordings. After the dogs became accustomed to the procedure they usually slept at intervals during the experiment.

Records of respiration were made at the same time that the intestinal movements were being recorded. The purpose of the respiratory tracing was not to study respiration only but to note the possible influence of respiration upon the intestinal movements. The respiratory movements were usually altered when the animal moved, which helped to explain unusual movements of the intestine.

A minimum of three days was allowed between the periods when an individual dog was used for different or repeated experiments. Thus, sufficient time was considered to have elapsed for the excretion of salts previously injected.

All of the experiments in type 2 were performed on unanesthetized dogs. An outstanding advantage of the method was that intestinal motility was not influenced by the anesthetic. Another advantage was the possibility of using the same dog for several experiments and studying the effect of different substances so that a comparison of reactions in the same animal could be made.

DISCUSSION

Two terms are used here to describe intestinal activity. The term intestinal motility or movement is used to refer to the

rapidly varying segmentations and peristaltic waves; the term intestinal tonus is used to refer to the slowly changing state of generalized contraction.

Movements of the intestine, such as localized segmentation, peristalsis and antiperistalsis, could not be identified individually by the methods used to record intestinal activity. Therefore, the general terms, motility or movement, were used to indicate segmentation or peristalsis of the intestine. Variations in intestinal tonus were easily recognized.

Lang's Solution.—Lang's solution was an effective agent in stimulating both intestinal tonus and motility. The first response was an increase in motility which was followed immediately by an increase in intestinal tonus. Intestinal motility appeared to be more easily stimulated than intestinal tone.

In the anesthetized animal Lang's solution seemed to exert its most pronounced action on the respiratory system. The smallest injection caused rapid and shallow breathing. Each injection produced a marked fall in blood pressure, although the heart continued to beat strongly.

In the unanesthetized dogs Lang's solution did not produce undesirable reactions upon the respiratory or circulatory systems. However, much smaller amounts of solution were administered in these cases than were given to the anesthetized dogs.

In none of the dogs, either anesthetized or unanesthetized, did the induced intestinal activity produce defecation. In general, Lang's solution produced strong intestinal activity although in some cases rather long intervals elapsed between the administration of the Lang's solution and the reaction.

Lang's solution consists of 60 Gm. (2 oz.) each of sodium chloride and sodium citrate dissolved in one quart of water.

Sodium Chloride.—Solutions of sodium chloride varying in strength from 2.5 per cent to 12.5 per cent were used. Each of the seven experiments with sodium chloride gave approximately the same results. Intestinal motility and tonus were stimulated by each of the different strengths administered. The use of different concentrations produced responses varying only in proportion to the amount of salt injected. The total amount of salt rather than the percentage strength of the solution injected appeared to be the factor influencing intestinal activity.

An early effect, more noticeable following the administration of the dilute rather than the stronger solutions of sodium chloride, was the stimulation of intestinal contractions without an increase in tonus. After additional saline had been injected, intestinal tonus was stimulated as strongly as intestinal contractions. Large amounts of saline produced extreme increases of intestinal tonus accompanied by suppression of motility. It appeared that the movements of the intestine, such as rhythmic segmentations and peristaltic rushes, possessed a lower threshold value than did the mechanism for increasing intestinal tonus.

Intravenous injections of solutions of sodium chloride were relatively nontoxic. Neither the circulatory nor respiratory systems were particularly susceptible to sodium chloride even though all the solutions were hypertonic. Hewitt, Greenwood and Nelson[7] showed that 60.6 Gm. (2 oz.) of sodium chloride in 30 per cent solution injected intravenously over a period of 17 minutes proved fatal to a dog weighing 18.18 kg. (40 lbs.). This was equivalent to 3.3 Gm. (.11 oz.) per kilogram of body weight. They concluded that the toxicity of sodium chloride was effected in three ways: 1) The osmotic effect, which increased blood volume and hence blood pressure; 2) a direct effect upon the medullary centers, at first stimulating and later paralyzing these centers; and 3) increasing the permeability of the cell membranes, causing fluid to escape more readily into the tissue spaces.

Sodium Citrate.—The response of the intestine to injections of sodium citrate was the opposite to that caused by sodium chloride. The citrate produced an increase of intestinal tonus with smaller amounts of solution than were needed to stimulate intestinal movements. It would appear that tonus was more susceptible to injections of sodium citrate than was the mechanism initiating intestinal movements. Intravenous injections of sodium citrate were relatively

toxic to both the circulatory and the respiratory systems.

With the intention of noting possible differences in the stimulating actions of the citrate ion and the chloride ion, solutions of citric acid and hydrogen chloride gas were injected intravenously into different anesthetized dogs. The results of administering a solution of hydrogen chloride gas to 4,500 indicated that the chloride ion did not stimulate intestinal activity. Similarly, a 5 per cent solution of citric acid was injected into each of three anesthetized dogs and in no case was intestinal motility or tonus stimulated.

By a process of elimination the citrate and the chloride ions did not seem to be the factors concerned in stimulating intestinal activity. Only the sodium ion remained to account for the stimulation of the intestine produced by sodium chloride and sodium citrate. The manner in which the sodium ions exerted their influence was not definitely revealed. Hammett[4] and Hammett and Nowrey[5] suggested that the sodium ion might act by increasing the permeability of the tissue to some other agent initiating the response. Hughson and Scarff[8] concluded that the sodium chloride had a direct effect upon the muscle fibers of the intestinal wall.

Sodium Bicarbonate.—A 5 per cent solution of sodium bicarbonate stimulated the small intestine of the dog more effectively than any of the solutions injected. Strong movements of the intestine were continued longer under the influence of sodium bicarbonate than any other salt. Comparatively large injections of sodium bicarbonate stimulated intestinal tonus so intensely that localized movements of the intestine were inhibited.

No reaction was elicited from the respiratory or circulatory systems by intravenous injections of moderate amounts of sodium bicarbonate. Over 600 cc. (20 oz.) of a 5 per cent solution was injected into one animal without producing an outstanding reaction.

Calcium Chloride.—Calcium chloride in 1 per cent solution did not stimulate intestinal activity as much as did the sodium salts. However, the influence of the injection seemed to persist longer with calcium chloride than with the sodium salts.

There has been considerable difference of opinion regarding the effect of calcium chloride upon intestinal movements.

Calcium Gluconate.—The intravenous injection a 5 per cent solution of calcium gluconate stimulated the small intestine more than did calcium chloride. Calcium gluconate did not affect the respiratory or circulatory system to any noticeable extent.

Calcium gluconate is soluble to the extent of about 3 per cent in 100 cc. (3.3 oz.) of distilled water, but by the addition of boric acid the solubility can be increased to 20 and even 30 per cent. The solution used in the experiments with calcium gluconate was stabilized with 4 per cent boric acid. In order to determine the effect of boric acid on intestinal motility, a 4 per cent solution of boric acid was administered to an anesthetized dog. Neither intestinal motility nor tonus was influenced by the injections. The respiratory and circulatory systems were not significantly altered by the injections. Thus, the results obtained from injecting calcium gluconate suspended in a solution by the aid of boric acid were considered to be due to the calcium salt and not to the boric acid.

Potassium Bicarbonate and Potassium Chloride.—Intestinal activity was inhibited in the experiments involving the intravenous injection of 5 per cent solutions of potassium bicarbonate and potassium chloride. These results were in agreement with Hazard and Wurmser[6] and Melnikov.[10] On the other hand, In[9] and Constantini and Ballarin[3] found that potassium chloride given intravenously stimulated intestinal movements. Considerable difference of opinion is evidenced in the literature in regard to the effect of potassium chloride on the intestine.

SUMMARY

The technic employed in experiments of type 1 permitted a study of the effects of intravenous injections upon the respiratory and circulatory systems as well as the intestinal musculature. This technic allowed a careful study of variations in intestinal

tonus and motility that occurred in a representative portion of the intestine. This method possessed the obvious disadvantage of being performed on an anesthetized dog by surgical interference.

Experiments of type 2 were performed on unanesthetized dogs that led a normal existence before, during and after the experiment. In this method the variations in blood pressure were not studied and the toxicity of an injected substance could be measured only by ordinary clinical methods.

By employing both types of procedure and comparing the results of each method, relatively inclusive data should be accumulated because the disadvantages of one method were nullified by the advantages of the other method.

A total of 30 experiments were performed. In type 1, 21 dogs were anesthetized and subjected to the experimental procedure. In type 2, nine experiments were performed with five unanesthetized dogs.

CONCLUSIONS

Lang's solution is an effective agent in stimulating both intestinal tonus and motility. However, it is slightly toxic to the respiratory and circulatory systems of the anesthetized dogs.

Solutions of sodium chloride stimulate the intestinal musculature. The first injections of small amounts stimulate intestinal motility primarily. Subsequent injections of larger amounts of sodium chloride solution stimulate intestinal tonus but suppress motility somewhat.

The quantity of sodium chloride injected rather than the percentage strength of the solution appears to be the factor stimulating the intestinal musculature. The response of the intestine varies in proportion to the amount of salt injected.

Large amounts of hypertonic solutions of sodium chloride administered intravenously are relatively nontoxic to the circulatory and respiratory systems.

An increase in tonus was the first and most persistent response of the intestine to the intravenous injection of sodium citrate. Intestinal motility is stimulated moderately by the citrate. A bradycardia and slowing of respiration result from large injections of sodium citrate.

The sodium ion appears to be instrumental in producing a stimulation of intestinal musculature. Sodium chloride and sodium citrate both stimulate the intestine but citric acid and a solution of hydrogen chloride gas do not affect the intestine. Therefore, it appears that the citrate and chloride ions do not stimulate the intestine. The sodium ion appears to be the only remaining factor and seems to be responsible for the stimulation of the intestinal musculature. The method whereby the sodium ion may be effective is not understood.

Lang's solution appears to stimulate the intestinal musculature by virtue of the sodium ions present.

Sodium bicarbonate stimulated the intestinal tonus and motility more effectively than any of the solutions used. The bicarbonate produced little or no effect on the respiratory and circulatory systems.

Calcium chloride did not stimulate the intestine as much as did the sodium salts but the effects persisted longer. Considerable danger of heart block accompanies the intravenous injection of calcium chloride.

Calcium gluconate stimulated the intestinal musculature more than calcium chloride. Boric acid, which was used to increase the solubility of calcium gluconate, apparently had no effect on the intestine.

The respiratory and circulatory systems were not altered significantly by the intravenous injection of calcium gluconate.

Potassium chloride and potassium bicarbonate appeared to depress the intestinal musculature. Potassium chloride was exceedingly toxic to the heart.

References

[1]Biebl, M.: Graphische darstellung der darmbewegungen an der in einen hautschlauch exventerierten darmschlinge. Klin. Wochnschr., ix (1930), part 2, pp. 1674-1675.

[2]Bors, E., and Polano, H.: Methodisches zur registierung von darmbewegungen. Arch. f. Exp. Path. u. Phar., clxvii (1932), pp. 238-243.

[3]Constantini, A., and Ballarin, G.: The action of various salts introduced by the intravenous route on intestinal peristalsis (title trans.). Boll. Soc. ital. biol. sper., ix (1934), pp. 1029-1932. Abst. in Chem. Absts., xxix (1935), p. 1882.

[4]Hammett, F. S.: The rôle of the change in hydro-

gen-ion concentration in the motor activities of the intestine. Amer. Jour. Physiol., lx (1922), pp. 52-58.

[5]Hammett, F. S., and Nowrey, Jr., J. E.: The rôle of the sodium and carbonate ions and the change in the sodium-calcium ratio in the contraction of the isolated duodenal segment of the albino rat. Amer. Jour. Physiol., lx (1922), pp. 48-51.

[6]Hazard, R., and Wurmser, L.: Action of potassium on the intestine (title trans.). Compt. Rend. Soc. Biol., cxxviii (1938), pp. 491-493. Abst. in Brit. Chem. & Physiol. Absts. A., cxi. Physiology and Biochemistry (1938), p. 914.

[7]Hewitt, E. A., Greenwood, D. A., and Nelson, V. E.: The effects of various sodium, potassium and calcium salts on respiration and blood pressure in the dog. Jour. A.V.M.A., xci (1937), n. s. 44 (5), pp. 555-570.

[8]Hughson, W., and Scarff, J. E.: The influence of intravenous sodium chloride on intestinal absorption and peristalsis. Johns Hopkins Hosp. Bul., xxxv (1924), pp. 197-201.

[9]In, K.: Influence of changes in the calcium and potassium ions concentration of the blood upon the intestinal and uterine movement *in situ*. I. Changes in intestinal and uterine movement by injections of calcium or potassium chloride (title trans.). Keijo Jour. Med., iv (1933), pp. 379-401. Abst. in Chem. Absts., xxviii (1934).

[10]Melnikov, G. P.: The influence of change of the blood composition upon the movements of the small intestine and their innervation (title trans.). Urachebnoie Delo., Nos. 6-8 (1923), pp. 128-130. Abst. in Physiol. Absts., viii (1923-24), pp. 247-248.

Heartworms

A study has been made on the survival and location of the microfilariae of *Dirofilaria immitis* in the dog.

An uninfected dog was injected intravenously with blood containing approximately 233,000 microfilariae. They survived in the blood stream of this dog for more than two years. None could be found, however, upon necropsy 2½ years after injection. No increase in the size of the larvae was noted. Comparatively few of the larvae appeared in the peripheral circulation after injection. Probably a part of the microfilariae are concentrated in the capillary networks throughout the body, and a part leave the blood stream altogether. There is some evidence that the macrophage system, activated by some type of immunological reaction, may destroy large numbers of microfilariae in a short time, but such reactions are of irregular occurrence and they do not regularly affect microfilarial longevity or periodicity. (*P. C. Underwood and P. D. Harwood. Survival and Location of the Microfilariae of Dirofilaria Immitis in the Dog. Journal of Parasitology, xxv, 1939, pp. 23-33.*)

Exanthematous Typhus in Cats

When fed upon or inoculated with typhus-infected material taken from guinea pigs, cats are capable of having an inapparent form of exanthematous typhus. The disease thus produced is not febrile, but the virus can be put in evidence in the brain of the cat 37 hours after the inoculation, only, however, to lose its pathogenicity after 69 days. The authors showed that three cats which had been in contact with human cases contracted the disease in inapparent form. (*Abst., Revue de Médecine Vétérinaire, xci, April 1939, p. 221.*)

Mustard Gas

The veterinary corps of nations at war need not be reminded of the terrors of mustard gas for man and animals. Mustard gas is thiodiglycol chloride. Its pet name in World War I was "Yperite," named for Ypres, where it was first used. The date was July 12, 1917. It differs from the other war gases in having a destructive external action in addition to its irritant effect on the respiratory tract. No systemic action has been observed. It disables and kills by its topical action. The fluid or its vapor in horses destroys the epithelial and subepithelial structure of the skin in large patches and, in sloughing, leaves a slow-healing wound.

Mustard gas disables horses in various ways. The damage is generally noticed about two weeks after exposure. Because battlefield horses are seldom exposed to high concentrations, respiratory troubles are not important. By the time animal-drawn trains arrive on the scene of an advance, much of the gas has been dissipated. It is contained mainly in holes cupped in the mud of roads and shellholes and, strangely, upon the leaves of trees, whence it settles down upon the backs of horses sheltered under them. Three weeks after a mustard-gas attack, horses lie in low places at the risk of having their sheaths slough off in great patches a month later. The gas derives its name from an odor resembling that of oil of mustard. It has no other claim to the name.

Designs for a Small Animal Hospital

ON PAGES 366 to 368 are reproduced the three small animal hospital designs selected as best among 26 entries in a contest sponsored by *The Veterinary Student*, official publication of the Iowa State College Student Chapter of the American Veterinary Medical Association.

Judges of the contest were Leonard Wolf, assistant professor of architectural engineering at Iowa State College; C. H. Covault, director of clinics, Division of Veterinary Medicine, Iowa State College; and Wayne H. Riser and Robert D. Wall, both small animal practitioners of Des Moines, Iowa.

Charles E. Mauser and Carlyle Peterson divided first and second prizes, and Warren Kroger was awarded third prize. All three are students in architectural engineering at the College. Each of the hospitals would house 20 to 25 dogs and would cost $8,000 to $10,000 to build.

Construction Outlines

DESIGN OF CHARLES E. MAUSER (SEE PAGE 366)

Walls.—The walls will be of brick; the exterior and interior surfaces should be spaced about an inch apart. The exterior wall will be backed by 5-in. zinc-flash tile. The interior walls will be composed of the same kind of tile as the backing of the exterior wall.

Ceiling.—Hard-surfaced plaster, painted with washable paint to match the wall surface. Metal lath will be used.

Floors.—The reception and office floors will be concrete. The corridor will be marbleized tile in dark sienna. All of the other rooms will have a concrete-surfaced floor sufficiently smooth to permit thorough cleaning.

Doors.—Slab doors veneered with birch, lightly stained, and varnished.

Windows.—Steel sash, and clear glass, with the exception of opaque glass in the windows of the lavatories. The reception and office windows will have light buff venetian blinds.

Kennels.—Concrete with nonabsorbent surface. One steel-grilled door, bolted to concrete. The doors will open separately for each kennel.

Heating and Ventilation.—A combination oil burner and air conditioning unit. Included in the heating room will be an incinerator.

Plumbing.—All kennel wards will be provided with hose connections. Steam will be available for sterilization purposes. Drains for each ward, the examination, surgery, bath and clipping rooms.

Lighting.—The office, reception room, examination and surgery rooms will be equipped with indirect lighting units.

DESIGN OF CARLYLE PETERSON (SEE PAGE 367)

Foundations.—Concrete footings and wall to grade line. Waterproofing—membrane around basement walls. Basement floor—4-in. concrete slab.

Structure.—Exterior walls—4-in. face brick, 1-in. air space, with 5-in. glazed tile back-up. Interior partitions—4-in. glazed tile. Floor construction—reinforced concrete. Roof construction—open-web steel joists, 2-in. concrete slab, 5-ply tar and gravel roof covering.

Ceilings.—Plaster on metal lath.

Windows.—Projected steel.

Interior Doors.—Slab veneer, except French doors from hall to examination and surgery rooms.

Heating and Air Conditioning.—Oil-burning boiler. Air heated by hot water coils adjacent to fan. Cold well water used for cooling in warm weather. Forced air circulated to rooms by duct from fan in furnace room up through the corner of laboratory to plenum chamber over corridor from which most rooms receive air. Office and basement rooms receive air through ducts in basement. Cold air is withdrawn from rooms through baseboard registers.

DESIGN OF WARREN KROGER (SEE PAGE 368)

Exterior Walls.—To be of 10-in. tile; exterior finish, 1½-in. stucco; interior, zinc-flash tile. All interior trim to be omitted.

Floors.—To be of rib-tile-slab construction, with 2-in. finish slab of concrete, troweled smooth in kennels and working space. For reception room, office and lavatory, slab to be covered with patterned, colored asphalt tile or linoleum.

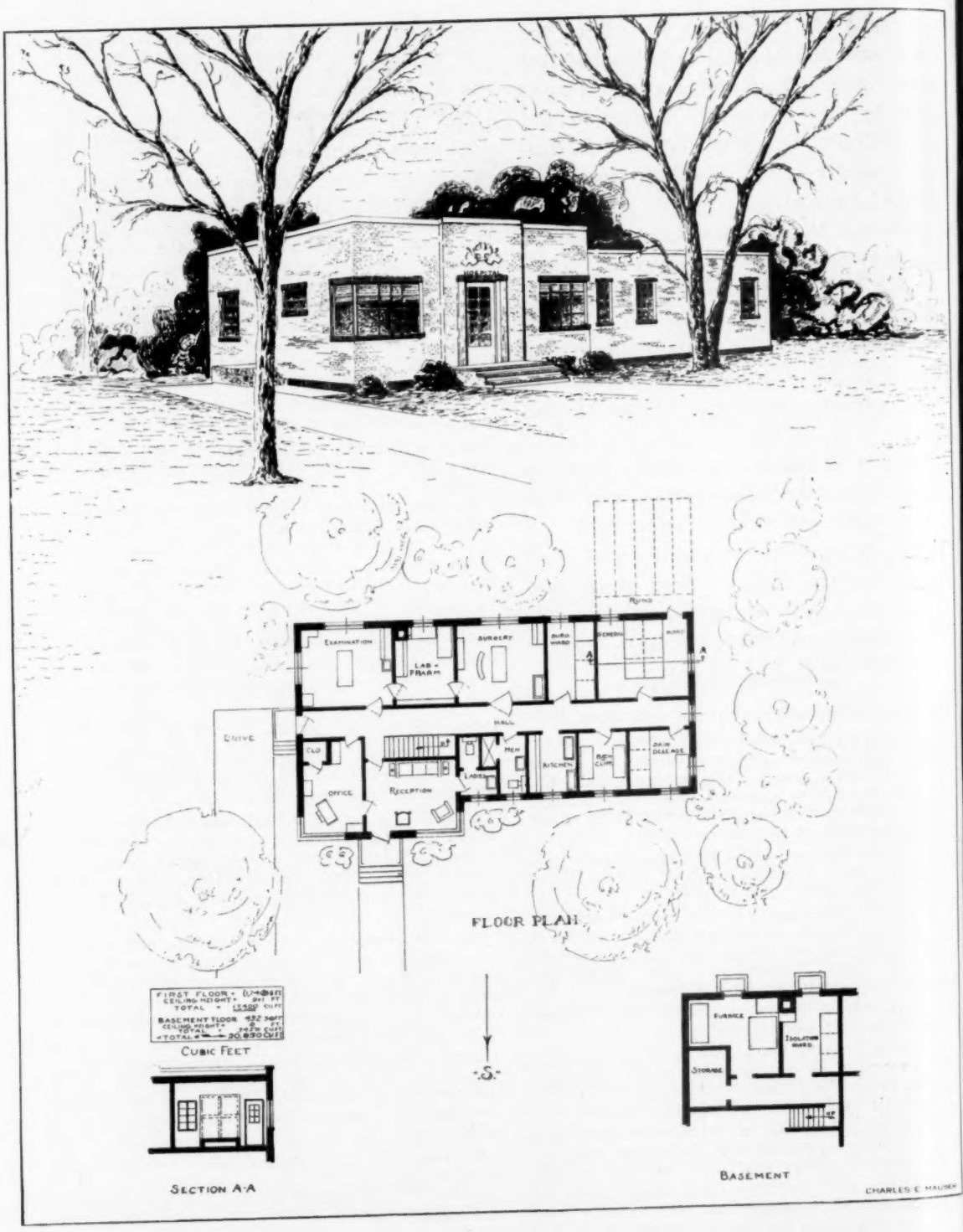
Interior Partitions.—To be of 5-in. zinc-flash tile. Surgery walls faced with glazed ceramic tile of desirable color, if budget permits. Otherwise, a good zinc-flash tile will give a satisfactory and inexpensive finish. All cabinets to be of noncorrosive metal.

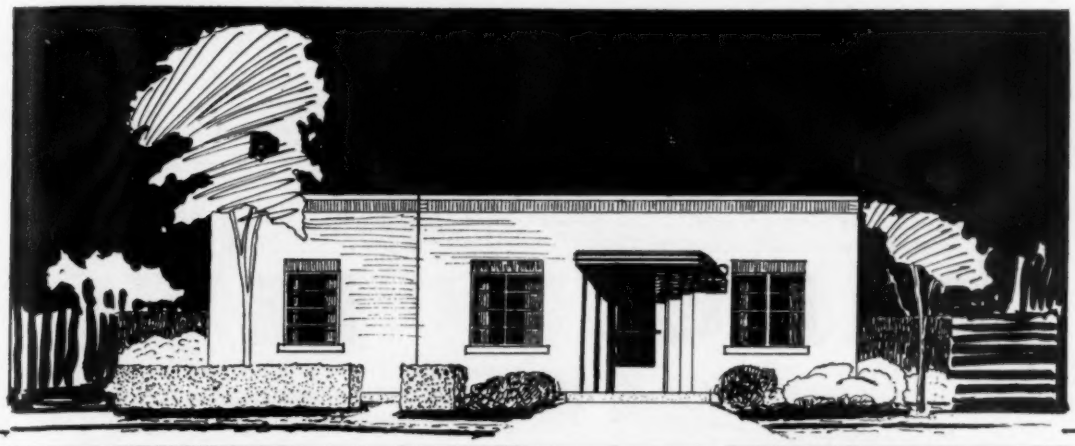
Roof.—To be durable and attractive in appearance. This can be obtained with any of the following materials: Asphalt or asbestos shingles, mingled-tone slate, or copper roof with raised seam.

Ceilings.—To be of hard plaster throughout, to facilitate cleaning.

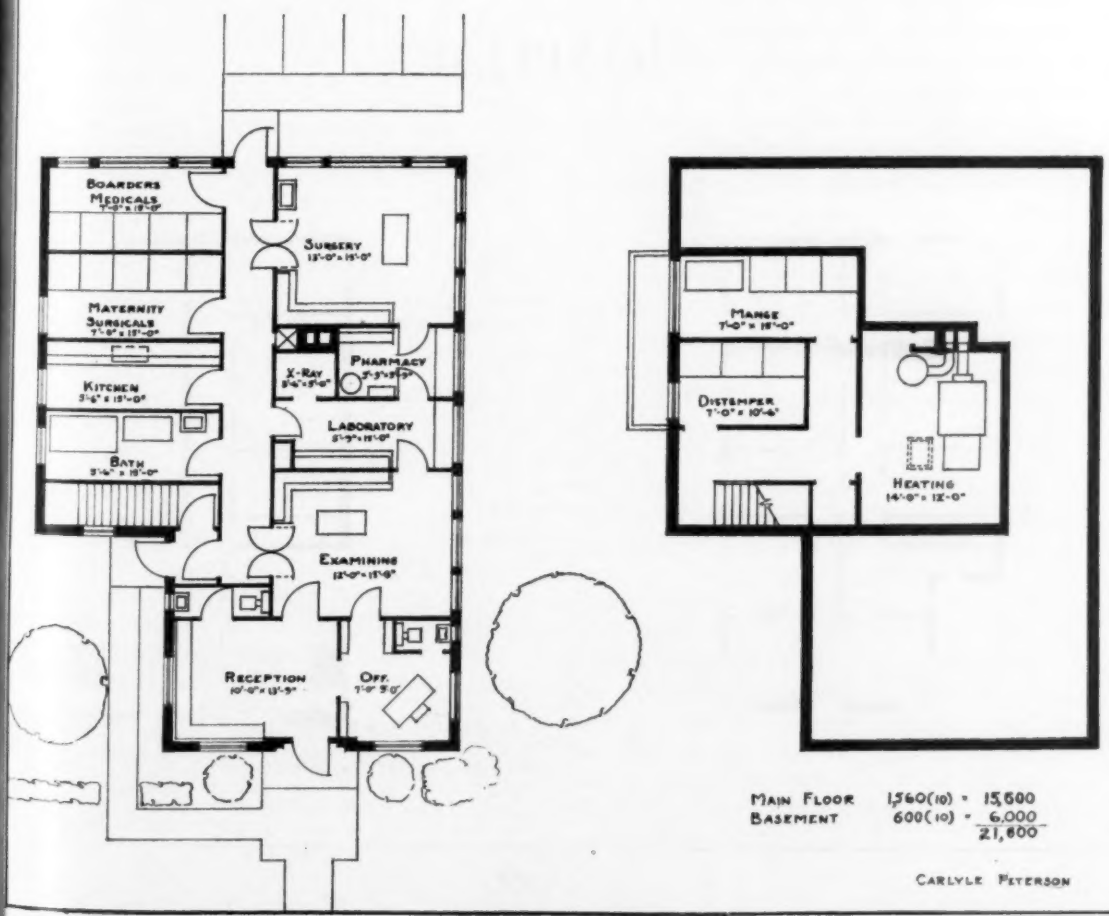
Doors.—To be slab type, finished with stain and varnish.

Windows.—To be of steel or wood sash, giving maximum light, sanitation and durability.


EXAMINATION
LAB - PHARM
SURGERY
DRIVE
OFFICE
RECEPTION
KITCHEN
FLOOR PLAN
CUBIC FEET
SECTION A-A
FURNACE
STORAGE
BASEMENT
CHARLES E. MAUSER

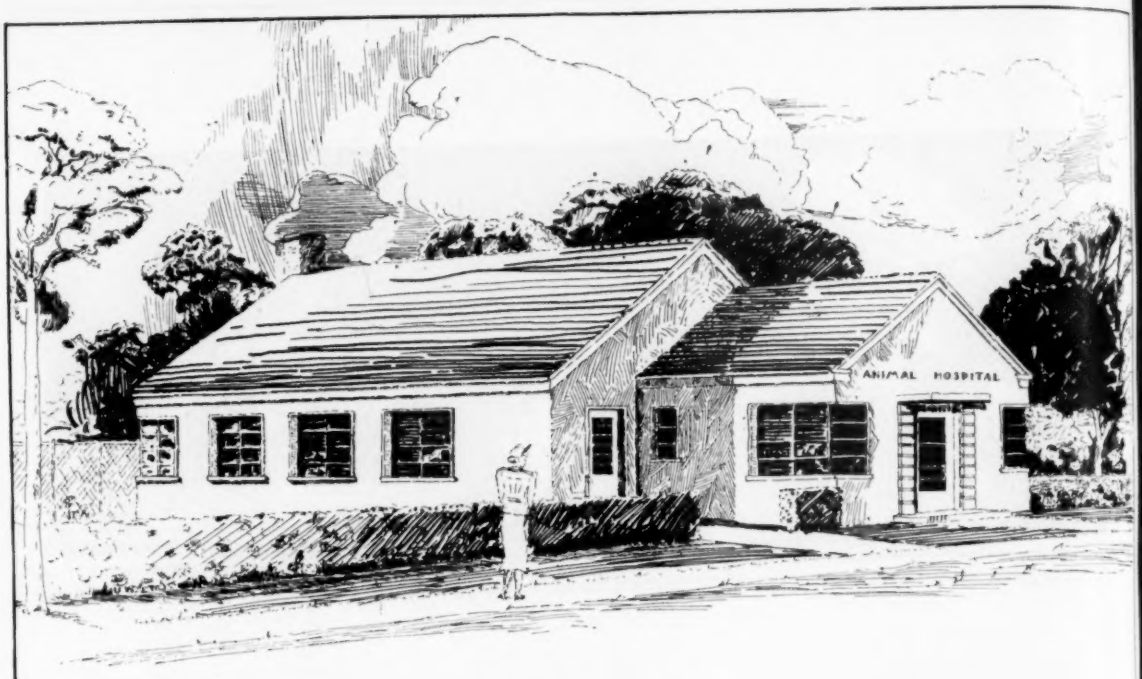


A SMALL ANIMAL HOSPITAL

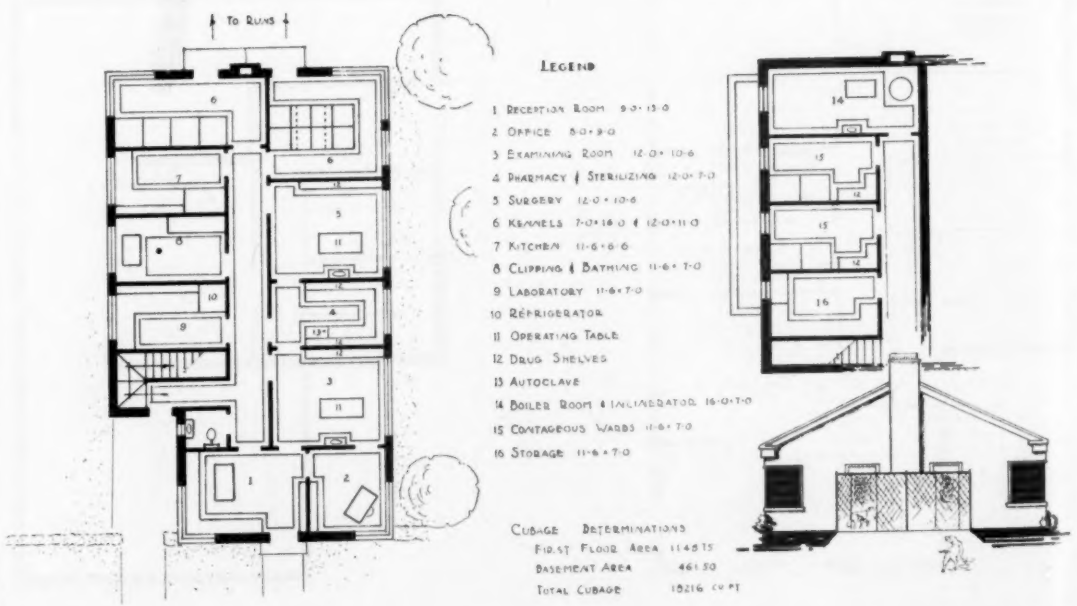


MAIN FLOOR	1,560(10)	= 15,600
BASEMENT	600(10)	= 6,000
		21,600

CARLYLE PETERSON



A Small-Animal Hospital



Incubating Hen's Egg as a Culture Medium for Brucella Abortus*

By H. J. METZGER, D.V.M., and FREIDA R. STOKES, M.S.
New Brunswick, N. J.

GOODPASTURE'S and Anderson's[1] report, in 1937, that *Brucella abortus* can be cultivated on the chorioallantoic membrane of the chick embryo led us to feel that this medium might be of some value in working with this organism. We have been working with this medium for over a year. This paper deals with two phases of our work: First, the effect of repeated egg-to-egg transfers on the virulence, cultural characteristics and antigenic properties of two different strains of *Br. abortus;* and second, an attempt to use the incubating egg for the purpose of typing different strains of this organism.

It has been generally accepted that animal passage does not alter the virulence of low-virulence or avirulent strains of *Br. abortus* which have been used for the preparation of vaccines. Huddleson,[2] in his discussion of the avirulent strain with which he was working, stated:

> There appears to be a lack of experimental evidence to substantiate the belief that an organism will reacquire its disease-producing properties once they have been completely lost. There may be such evidence in existence but diligent search has failed to produce it.

Cotton and Buck[3] stated:

> Whether low-virulence strains are reasonably stable in virulence and immunity-inducing effects is somewhat problematic. However, one strain that has been employed in our immunization experiments has appeared to remain fairly constant in virulence during the past three or four years, as indicated by repeated tests on guinea pigs.

Cotton and Buck inoculated pregnant cows with this strain, checking the virulence of the organism recovered from the calf or uterine exudate by guinea pig inoculation. The organism recovered from one cow was reinoculated into a second pregnant animal. Cultures from this second animal showed no increase in virulence when inoculated into a series of guinea pigs. They concluded:

> It thus appears that the danger that strains of *Br. abortus* which have lost much of their virulence, will regain it by long sojourns in cattle, may be slight.

In the work of Cotton and Buck, the organism apparently was recovered from one animal on artificial mediums before inoculation into another animal. We have used the incubating egg because it enables one to make frequent transfers from one living organism to another without the use of an artificial medium.

METHODS AND MATERIALS

United States bureau of animal industry strain 19 and a recently isolated strain C. G. were used throughout the experiment. Strain C. G. was isolated from the udder of a cow in a dairy in which no vaccination has been done. Guinea pigs inoculated with this strain show enlarged spleens, occasional abscessed testes, and a high blood titre when killed at six weeks.

All eggs were inoculated on the tenth day of incubation. They were inoculated through the natural air sac into the chorioallantoic membranes by a method which was developed by F. R. Beaudette of the New Jersey Agricultural Experiment Station. The eggs were incubated at a temperature of 102.5 to 103.0° F. The inoculated eggs were candled daily. All of those with dead embryos were removed to the refrigerator. All embryos, including the live ones, were cultured on potato agar on the seventh day after inoculation. The membranes, liver, spleen and brain were cultured. All cultures were checked by microscopic examination and also checked for agglutinability with positive and negative serum. One culture from each egg was incubated aerobically. All other cultures were incubated with 10 per cent carbon dioxide. A record

*Journal series paper of the New Jersey Agricultural Experiment Station, department of dairy husbandry. Presented before the Section on Research at the 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1939.

was kept of the lesions found in each embryo.

At the beginning of the experiment a series of guinea pigs and eggs were inoculated with graduated doses of strain 19 and another series of guinea pigs and eggs were inoculated in like manner with strain C. G. The guinea pigs were killed and cultured six weeks after inoculation. The lesions of the chick embryos and the guinea pigs and the blood titre of the guinea pigs were noted for purposes of comparison with similar inoculations to be made later in the experiment.

Two eggs from this first series which were inoculated with approximately 130,000 organisms of strain 19 and two eggs which received approximately the same number of strain C. G. were selected for the inoculation of another series of eggs. The inoculum was prepared by grinding the aseptically removed liver with sterile sand and washing the sand with 20 cc. (.66 oz.) of sterile saline. Each of two eggs was inoculated with 0.2 cc. (3 minims) of a 1:8 dilution of each 20-cc. suspension. With each transfer the egg living the longer period was selected for the inoculation of two more eggs. The number of organisms inoculated, with each transfer, were counted by plating on tryptose agar 0.1 cc. (1.5 minims) of a 1:10,000 dilution of the original 20-cc. suspension.

RESULTS

Suspensions of strain 19 and of strain C. G. from the eleventh transfer and from the original culture of these two strains were inoculated into guinea pigs, which were killed six weeks after inoculation. The lesions and blood titres of the guinea pigs and the cultural characteristics of the organisms recovered from the guinea pigs indicated that neither of these two strains had been changed by eleven egg-to-egg transfers. Strain 19 continued to grow aerobically throughout the experiment, and strain C. G. refused to grow without carbon dioxide. No rough colonies were observed in any of the plates examined throughout the experiment. Antigens produced from these two strains obtained from the 22nd transfer checked exactly with our stock antigen.

AN ATTEMPT TO TYPE DIFFERENT STRAINS

The possibility of using the incubating egg for the purpose of typing different strains of *Br. abortus* also was investigated by inoculating two series of eggs, one with different doses of strain 19 and the other with varying doses of strain C. G. The dosages varied from 150 to 13,000,000 organisms. The length of time the embryo lived after inoculation and the lesions produced were recorded. A careful study of these records indicates that there is no significant difference between the effects produced by strain 19 and those produced by strain C. G.

DISCUSSION

It is possible that the continued serial passage of *Br. abortus* through eggs may cause it to undergo certain changes. Gallavan,[4] working with *Haemophilus influenzae,* reported a marked increase in the ability of this organism to agglutinate with polyvalent horse serum following 20 serial passages through eggs. Gallavan and Goodpasture[5] reported that a similar number of egg-to-egg transfers of an *H. pertussis* caused slight changes in its cultural characteristics and a marked decrease in the agglutination titre of this organism. We are continuing the work in hope that we may be able to demonstrate some changes in these two strains.

The question also arises as to whether the results obtained could be duplicated with animals instead of incubating eggs. Our work seems to indicate that the chick embryo acts as a passive sort of medium which offers little or no resistance to the growth of *Br. abortus.* We have recovered this organism from the membranes, liver, spleen, and brain of chick embryos which were inoculated with approximately six organisms. We have been unable to demonstrate any antibodies in the blood serum of infected live embryos removed from the egg on the 20th day of incubation. There seems to be no difference in the response of the egg to inoculation with strains of either high or low virulence. It would be interesting to see this work carried on in

a similar way with guinea pigs instead of eggs.

SUMMARY

Eleven egg-to-egg transfers of two strains of *Brucella abortus*, strain 19 and a virulent strain C. G., failed to cause these strains to undergo any changes in virulence, cultural characteristics or antigenic properties.

Efforts to use the incubating egg for the purpose of typing different strains of *Br. abortus* were unsuccessful.

References

[1]Goodpasture, E. W., and Anderson, K.: Amer. Jour. Path., xiii (1937), p. 149.
[2]Huddleson, I. F.: Mich. Agr. Exp. Sta. Tech. Bul. 65 (1924).
[3]Cotton, W. E., and Buck, J. M.: Jour. A.V.M.A., lxxxiv (1934), n. s. 37 (3), p. 329.
[4]Gallavan, M.: Amer. Jour. Path., xiii (1937), p. 911.
[5]Gallavan, M., and Goodpasture, E. W.: Amer. Jour. Path., xiii (1937), p. 927.

Handicaps in Goat-Milk Production

In an article entitled "The Milk Goat Industry and Its Future," M. A. Emmerson of the School of Veterinary Medicine, University of Pennsylvania, tells the readers of *The Goat World* that while milking goats have been kept by certain families since the earliest days of American history, the superior value of goat's milk has only recently begun to attract general attention.

Reports of numerous instances of chronically sick persons having been restored to health by substituting goat's milk for cow's milk in their daily dietary are increasing the popularity of the domestic goat as an additional source of the nation's milk supply. The author is not convinced that pasteurized milk is as efficient as natural milk. On the contrary, "pasteurization, in some instances at least, is just a safe way to market a dirty milk," and furthermore, it "does reduce the palatability and nutritive value of good, clean milk."

An obstacle in the development of the goat-milk industry is the difficulty of procuring a regular supply of milk comparable with that of the cow-milk industry. In cities goat milk can not be obtained with the same ease as cow's milk, which is furnished by a highly organized industry, representing a tremendous investment, that is ever on guard to prevent the development of a competing industry.

Granted that goat's milk is all that is claimed for it, the author surveys the situation with the view of recommending means of overcoming the obstacles confronting the goat-milk industry, such as maintaining year-around production. Under present breeding conditions goats furnish abundant milk only during the second six months of the calendar year (in the northern hemisphere). Does do not ovulate actively during the first six months of the year and therefore prevent all-year production. To overcome this handicap by selective breeding appears to be an important preliminary step in the development of the goat-milk industry. Moreover, congenital deformities which are common in goats should be bred out. Needless to say, records of milk production are paramount in the breeding campaign.—*From The Goat World, xxiv, December 1939.*

[In his article* *Le Cycle Oestral Chez les Femelles Mammifères*, Berthelon says: "For some authors, the season for reproduction (in goats) is the fall (Stross) and for others there is a second season in the spring (Milovanov, Schmaltz). The number of cycles within these seasons is not well known. The duration of the estrual revolution is about the same as that of the ovine species (two or three weeks). Heat is, however, somewhat longer, persisting often two to three days. Ovulation occurs toward the end of the period and, to obtain the best results in artificial insemination, the second day of heat is chosen in preference to the first day (Milovanov). Heat in goats is manifested by restlessness and agitation, diminished appetite and modification of lactation. Boiling generally coagulates the milk (Stross)."]

Take care of thy business and thy business will take care of thee.—*Benjamin Franklin.*

*Recueil de Médecine Vétérinaire, cxv, February 1939, p. 79.

Indoor-Hen-Battery Mortality*

By M. W. EMMEL. D.V.M., M.S.
Gainesville, Fla.

UNDER indoor-hen-battery conditions, paralysis, leucemia and emaciation have constituted a major mortality problem, with losses ranging from 20 to 60 per cent of the hen-battery population. In many instances hen-battery-plant operators have entered upon this method as an ideal means of housing birds only to encounter high rates of mortality which have been responsible for a discontinuance of operations.

Previous papers[1-5] have shown that fowl paralysis and leucemia as well as a number of other manifestations occurring under the general classification of leucosis can be induced by bacteria of the paratyphoid group. Infection was associated with intestinal parasitism and was dependent upon the interrelationship of a number of factors. It has been shown that the microörganisms initiate the basic process of hemocytoblastosis, which, in turn, leads to the development of many of the manifestations.

Under indoor-hen-battery environments, the writer has been unable to demonstrate the association of microörganisms of the paratyphoid group and intestinal parasitism with the occurrence of paralysis and leucemia. On the other hand, an examination of the blood of hens confined in indoor-battery plants has shown that hemocytoblastosis occurs in all birds which have been housed in the plant more than a month.

A definite syndrome is associated with the occurrence of paralysis, leucemia and emaciation under indoor-hen-battery conditions. This syndrome consists of: Poor development of some birds particularly in evidence at maturity, irregular egg production and molting. Most poorly developed birds never begin egg production. Other birds become good producers and maintain production for varying periods, then suddenly stop permanently, while others resume spasmodic production. Paralysis, leucemia and emaciation begin to occur under indoor-hen-battery conditions when the birds are 4 to 6 months of age.

The condition in which birds show progressive emaciation followed by death has been diagnosed as chronic hemocytoblastosis. The comb becomes flaccid, shriveled, somewhat pale, scaly and slightly grayish in color. The droppings are scant and often whitish green in color. Emaciation is progressive over a period of three to eight weeks. The bird becomes anemic, with a low erythrocyte count and a low hemoglobin reading. Hemocytoblastosis, characterized by a high percentage of vacuolar lymphocytes, occurs. At autopsy the liver, spleen, intestine and kidneys are atrophied; hematopoietic tissue is degenerative and often exhausted. The smallest liver found in an adult bird affected with chronic hemocytoblastosis weighed 11 Gm. (.35 oz.) as compared with livers weighing 40 to 53 Gm. (1.3 to 1.8 oz.) in normal birds confined under controlled conditions.

It has been found that adverse atmospheric conditions as related to inadequate ventilation and excess populations are responsible for the development of paralysis, leucemia, chronic hemocytoblastosis and the accompanying syndrome under indoor-hen-battery conditions. Table I gives the summary of a comparison of indoor-housing experiments under poor (closed) and fair (open) conditions, and of outdoor conditions throughout a period of ten months, Barred Plymouth Rocks serving as the experimental subjects. A total of 125 birds were included in each group, the experiment being conducted in two trials. Under closed conditions of ventilation the mortality from paralysis, leucemia and chronic hemocytoblastosis amounted to 32 per cent; under fair conditions of ventilation the

*From the Florida Agricultural Experiment Station. Presented before the Section on Poultry at the 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1939.

TABLE I—Summary of mortality in experiments in which Barred Plymouth Rock chickens were housed indoors versus outdoors for a period of ten months.

GROUPS	TRIAL	NO. BIRDS	MORTALITY				
			PARALYSIS	LEUCEMIA	CHRONIC HEMOCYTOBLASTOSIS	OTHER CAUSES	TOTAL
I	I	60	4	8	7	1	20
Indoor (closed)	II	65	3	7	11	2	23
II	I	60	3	2	6	—	11
Indoor (open)	II	65	3	3	4	1	11
III	I	60	—	—	—	1	1
Outdoors	II	65	—	—	—	3	3

mortality was 16.8 per cent. None of the control birds died of these manifestations.

Table II gives a summary of experiments in which Rhode Island Red pullets were reared outdoors for six months, then moved to indoor hen batteries. Four different trials were conducted, a total of 93 birds being used. The 21 control birds were kept outdoors throughout the experiments. After being transferred to indoor hen batteries, 41.1 per cent of the 93 birds developed fowl paralysis, leucemia or chronic hemocytoblastosis during the eight months they remained indoors, whereas none of the control birds developed any of these manifestations.

A study of four outdoor-hen-battery plants which housed 200, 600, 800, and 1,200 birds, respectively, showed that no deaths from paralysis, leucemia or chronic hemocytoblastosis occurred.

In another experiment extending over a period of three years, a varying number of Barred Plymouth Rock chickens were reared and housed in batteries during each successive one-year period. During the first year a population of 75 was reduced to 42. During the second year a population of 100 was reduced to 41. During the third year only one of 37 birds died; this bird died during the first month of the experiment. The writer believes that each indoor-hen-battery plant has a rather definite biological saturation point, *i. e.*, a rather definite number of birds can be kept successfully under the environmental conditions surrounding each indoor-battery plant. Should populations be maintained

TABLE II—Summary of mortality in experiments in which Rhode Island Red pullets were reared outdoors, then moved to indoor batteries in a room in which ventilation was considered inadequate.

TRIALS	NO. BIRDS	MORTALITY				
		PARALYSIS	LEUCEMIA	CHRONIC HEMOCYTOBLASTOSIS	MORE THAN ONE MANIFESTATION*	TOTAL
I	12	—	—	3	—	3
II	31	2	5	8	3	15
III	27	3	6	6	2	13
IV	23	1	3	4	—	8
Controls (outdoors) V	21	—	—	—	—	—

*Diagnosed as first manifestation shown.

in excess of this point, losses from paralysis, leucemia and chronic hemocytoblastosis associated with the syndrome previously described will occur.

In case the operator makes a practice of replacing birds lost through mortality, he is maintaining adverse atmospheric conditions which make the mortality problem a continuous one.

Experiments are being conducted to determine the factors involved in adverse atmospheric conditions. Under adverse atmosperic conditions, metabolism is apparently altered with the result that an autointoxication occurs which is evidenced by the development of hemocytoblastosis. If birds are raised under battery conditions, paralysis, leucemia and chronic hemocytoblastosis appear when the birds are about 4 months old. Consequently, the development of these manifestations is a chronic process.

Besides being of importance as far as indoor-hen-battery mortality is concerned, adverse atmospheric conditions have other practical applications. In battery broiler and fryer plants the development of hemocytoblastosis as a result of adverse atmospheric conditions leads to reduced resistance, retarded growth, poor feathering and lack of uniform growth. For these reasons adverse atmospheric conditions are of vital importance during the brooding period and are of concern not only in the brooder itself but also in the room in which the brooder or brooders are housed. Birds are not held in battery broiler and fryer plants long enough for paralysis, leucemia and chronic hemocytoblastosis to occur.

Unpublished data show that artificially hatched chicks have marked hemocytoblastosis as compared with hen-hatched chicks. Adverse atmospheric conditions as they relate to respiration are responsible in part at least for the occurrence of hemocytoblastosis in the developing embryo, which results in a day-old chick of reduced vitality.

Lastly, adverse atmospheric conditions should be of vital concern to every investigator conducting research on the diseases classified under the general term "leucosis." Various groups of birds in transmission experiments have shown from 10 to 33 per cent greater susceptibility to the transmissible diseases of this group when raised under adverse atmospheric conditions than when similarly caged outdoors. Hemocytoblastosis-free experimental birds have not been of great concern to previous investigators.

The experiments reported herein and the application of adverse atmospheric conditions to the various phases of poultry production and management have arisen through difficulties encountered by the author in attempts to raise hemocytoblastosis-free experimental birds. It is believed that the wide variations in the results of transmission experiments reported by various investigators are in part due to the problem of adverse atmospheric conditions as discussed in this paper.

References

[1]Emmel, M. W.: The etiology of fowl paralysis, leukemia and allied conditions in animals. I. Introduction. History and a bacterial theory of the etiology of these diseases. II. The intravenous injection of suspensions of *Salmonella aertrycke* in the chicken. Fla. Agr. Exp. Sta. Bul. 284 (1935).

[2]*Idem:* The etiology of fowl paralysis, leukemia and allied conditions in animals. III. The intestinal flora of chickens affected with enteritis associated with intestinal parasitism. IV. The pathologic manifestations of the causal micro-organisms in the fowl. Fla. Agr. Exp. Sta. Bul. 293 (1936).

[3]*Idem:* The etiology of fowl paralysis, leukemia and allied conditions in animals. V. The oral exposure of chickens infected with various species of coccidia to *Salmonella aertrycke.* VI. The oral exposure of chickens infested with Ascaridia, Taenia and Capillaria to *Salmonella aertrycke.* Fla. Agr. Exp. Sta. Bul. 305 (1936).

[4]*Idem:* The etiology of fowl paralysis, leukemia and allied conditions in animals. VII. Hemocytoblastosis in chickens as induced by *Salmonella aertrycke.* VIII. Hemocytoblastosis in naturally occurring cases of fowl paralysis, lymphomatosis and in other birds from the same flocks. Fla. Agr. Exp. Sta. Bul. 306 (1936).

[5]*Idem:* The etiology of fowl paralysis, leukemia and allied conditions in animals. IX. *Salmonella enteritidis* as a causal micro-organism for this group of diseases in the chicken. X. *Salmonella schottmülleri* as a causal micro-organism for this group of diseases in the chicken. Fla. Agr. Exp. Sta. Bul. 313 (1937).

Martin (*Poultry Practice,* p. 5) points out that, in 1936, 63,982,000 chickens out of a total of 420,257,000 died on the farms of the United States. The direct money loss was $48,000,000 for adult mortality alone. To this tremendous sum, the author adds indefinite figures for overhead costs.

Wheat Germ Oil in Fowl Paralysis*

By E. P. JOHNSON, D.V.M., M.S., Ph.D.
Blacksburg, Va.

THE RECENT reports by Butler and Warren[1] and by Butler, Warren and Hammersland,[2] concluding from field observations that wheat germ oil has both prophylactic and therapeutic value in fowl leucosis, including paralysis, has stimulated widespread hope in the future control of this disease. The conclusions reached by these workers appear especially significant, since Adamstone[3] reported the occurrence of "lymphoblastoma" in young birds kept on a diet which had been treated with ferric chloride to destroy vitamin E. It is needless to say that these results, embracing a problem of such magnitude, should stimulate further research in an attempt to discover further possibilities in this field.

Since the experiments on which this report is based were undertaken, however, reports by other workers on this subject have become available and brief mention of their results should be made before a discussion of these experiments is presented.

Davidson and Schaible[4] added 5 per cent wheat germ meal to the laying ration, but found no difference in the incidence of leucosis among treated and untreated lots. Cole[5] likewise failed to notice any beneficial results in the treatment of 31 chickens affected with fowl paralysis under controlled laboratory conditions. Jungherr[6] reported negative results and concluded that there is no valid reason to assume a specific antileucotic property of wheat germ oil. In addition, Taylor and De Ome[7] found that the feeding of wheat germ oil had no appreciable effect on the incidence, type, or age onset of lymphomatosis.

EXPERIMENTAL PROCEDURE

Between the dates January 10 and February 12, 1938, seven birds with varying degrees of leg paralysis were placed in individual cages and treated twice weekly with 1 cc. (15 minims) each intramuscularly of a good grade of cold-pressed wheat germ oil. This treatment was continued for two months in all cases that did not die previous to the expiration of this period. At death, or soon after the termination of the treatment, the birds were autopsied and both gross and microscopic examinations were made for lesions.

Two of the birds died 19 and 62 days, respectively, following the first injection. At autopsy one bird was blind in one eye, had marked enlargement of one sciatic nerve, and the liver was at least twice the normal size. Microscopically, typical areas of cell proliferation were present in the involved organs. The other bird had similar lesions in the sciatic nerve of one leg and in the liver. The eyes, however, were not involved. No improvement was noted in the other five. Two became gradually worse, and all had typical gross and microscopic lesions of leucosis at the time of autopsy. An extensive area of fibrosis developed in the injected area of the pectoral muscles of one.

These results were 100 per cent negative as far as curative value of the wheat germ oil was concerned. We then decided to determine what the results would be from a prophylactic point of view. For these tests 27 chicks hatched in our laboratory and brooded in previously sterilized cages were divided into three groups. One bird was destroyed because it developed perosis or slipped tendons. Of the remaining 26, six served as controls, and two groups of ten birds each were injected intravenously when 6 weeks of age with 0.5 cc. (7 minims) of saline suspension of spleen taken from an advanced case of leucosis with symptoms of leg paralysis. One of these groups of ten chicks received 0.5 cc. each of a good grade of cold-pressed wheat germ oil in their feed three times weekly, starting one month prior to the time of injecting the leucotic material. Soon after the injections the oil was increased to 1 cc. per bird. Wheat

*From the Virginia Agricultural Experiment Station.

germ oil was fed in this manner for a period of nine months and the results with the three groups of birds were carefully observed. During this period three of the ten birds receiving the oil developed a form of leucosis. Symptoms first appeared three, five and eight months, respectively, after the birds received the injections of leucotic material.

Two of the ten birds injected, which did not receive wheat germ oil, developed the disease. One of these developed symptoms of leg paralysis two months following the injection and the other died after four months. At autopsy the latter had a greatly enlarged liver and spleen, as well as an ovarian tumor.

At the termination of the experiment, or about nine months after the infective material was administered to the two groups, the six control birds also were killed and autopsied. All of these were normal, with the exception of one. This bird developed a secondary anemia; the spleen was enlarged and contained focal areas of cell proliferations that were suggestive of leucosis.

DISCUSSION

Without going into detailed discussion as to the characteristic lesions produced in leucotic diseases of fowls and those produced in vitamin deficiencies, it is necessary only to mention that leucotic diseases are characterized, in general, by ectopic proliferation of hematopoietic elements, while vitamin E deficiencies, like other avitaminoses, are primarily characterized by degenerative processes of selected tissues. In view of these differences, as concluded by Jungherr,[6] there is no pathological ground for assuming a specific antileucotic property of wheat germ oil. The results of the writer's experiments confirm these conclusions.

References

[1]Butler, W. J., and Warren, D. M.: Fowl leukemia and vitamin E, a preliminary report. Jour. A.V.M.A., xcii (1938), n. s. 45 (2), pp. 204-206.

[2]Butler, W. J., Warren, D. M., and Hammersland, H. L.: Nutrition as a factor in the incidence of fowl leukosis. Jour. A.V.M.A., xciii (1938), n. s. 46 (5), pp. 307-315.

[3]Adamstone, F. B.: A lymphoblastoma occurring in young chicks reared on a diet treated with ferric chloride to destroy vitamin E. Amer. Jour. Cancer, xxviii (1936), pp. 540-549.

[4]Davidson, J. A., and Schaible, P. J.: Pullet mortality. Some observations on the effect of adding wheat germ meal to the laying ration. Mich. Agr. Exp. Sta. Quart. Bul., xx (1938), 3, pp. 1-6.

[5]Cole, R. K.: Vitamin E and avian neurolymphomatosis. Sci., lxxxviii (1938), pp. 286-287.

[6]Jungherr, E.: Wheat germ oil in the control of fowl paralysis and kindred diseases. Proc. 42nd Ann. Meet. U. S. Live Stock Sanitary Assn. (1938), pp. 171-180.

[7]Taylor, L. W., and DeOme, K. B.: Failure of wheat germ oil to prevent lymphomatosis in chickens. Jour. A.V.M.A., xcv (July 1939), 748, pp. 73-76.

Danger Lurks in Luke Warm Linseed Cake

The cyanogenic properties of linseed cake are acknowledged, but its aptitude to form hydrocyanic acid in toxic amounts when fed warm has not been emphasized, except by authors who have made a systematic study of the subject. The consensus among these is that the cyanogenic glucoside of these products, linamarine, in the presence of luke warm water, through the action of a specific diastase known as linase, elaborates glucose, acetone and hydrocyanic acid. The scientific proof of this action is confirmed by the observation of veterinarians to the effect that linseed cake is dangerous when given to animals mixed with tepid water.

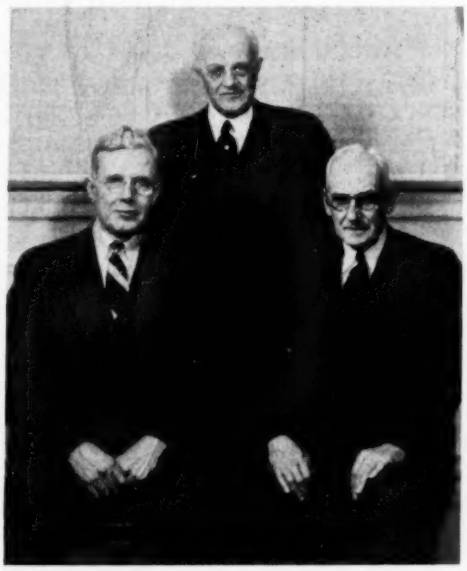
Linase is a specific diastase that is inert at 60° C. Ordinary boiling does not prevent it from becoming cyanogenic when the temperature again drops to luke warm degree, and when boiled for a long while, the resulting pulp is not at all appetizing. Hogs, horses, cattle and sheep refuse to eat it.

The author proposes that the factory expressing the oil should destroy the linamarine, which is not particularly difficult to do, in order to prevent all toxic accidents of this type, for, as long as linseed cake is allowed to retain its linamarine, such accidents will continue to occur. The ferments of the small intestine are capable of elaborating hydrocyanic acid from cake containing that component. *(Jean Isnard. What Is the Best Means of Feeding Cyanogenic Linseed Cake? [Title translated.] Revue de Médecine Vétérinaire, xci, April 1939, pp. 212-216.)*

What the Practitioner Can Best Do for the A.V.M.A.*

By CASSIUS WAY, D.V.M.
New York, N. Y.

IT IS A privilege for me to meet with you here today. The invitation is one which I deeply appreciate. Illinois always has been identified in my mind with the best corn and the best crops, all grown on the richest, blackest soil in the Mississippi Valley. In fact, coming to the grand state of Illinois is like coming home for me. Illinois has a warm spot in my heart because it gave me my first insight into the realities of rural veterinary service. I spent some of the happiest years of my life as a dairy inspector for the Borden Company and a "cub veterinarian," living in McHenry county. One winter I distinctly remember driving on runners for 13 consecutive weeks without seeing a wheeled vehicle. Never had I seen such fine dairy farms and I had never seen such big red barns as I found in Illinois.



Board of Governors of the American Veterinary Medical Association (left to right): Cassius Way, New York City; H. W. Jakeman, chairman of the Board, Boston, Mass.; and A. E. Wight, Washington, D. C.

I shall always cherish the memory of having been intimately acquainted with such outstanding men as Baker, Hughes, McKillip, Quitman, White and Wright, all pioneers in veterinary medicine in Illinois.

THE MILK OF FORMER DAYS

I have said that I learned realities out here. I am sure you will agree with me when I tell you that it was in Illinois that I learned about the dilution of milk. In case you do not know, in these days of buying milk on butterfat and bacterial standards, it was then a custom to put one can cover of water to the can of milk. During the months of short production the amount was increased to two can covers. And, just to prove that my early education in Illinois was practical and broad, I might say that I have not forgotten how to test Holstein milk accurately. The method is to drop a silver dollar in the can of milk; if you can read "In God We Trust" on the dollar, it is Holstein milk.

ILLINOIS ASSOCIATION CONGRATULATED

But, with all jokes cast aside regarding the good old Holstein cow — for, after all, she is about the most profitable of all the milk-producing breeds —permit me to congratulate you most sincerely on the splendid program you have for your annual meeting. I was pleased to see that you have such men as Carpenter, Rinehart, Anderes and Boynton listed as speakers. I am confident that this meeting will be outstanding among the many annual meetings of the different state veterinary medical associations this winter.

I know that many of you men have long been members of the American Veterinary Medical Association. You will, therefore, be as familiar as I with the fact that the A.V.M.A. heartily endorses the idea back of these state meetings—that of keeping us practicing veterinarians informed and up-to-date on the new developments in our profession. I would like to speak for a few minutes regarding progress in two or three fields having special bearing on A.V.M.A. policies and activity.

*Presented before the 58th annual meeting of the Illinois State Veterinary Medical Association, Springfield, Ill., February 15-16, 1940.

From the day 32 years ago when I received a diploma from the president of Cornell University, I have been impressed by the fact that our educational institutions and veterinary colleges are the very backbone of the profession in the United States. It is the responsibility of the colleges not only to educate the undergraduates but to reëducate the graduates who are out in the field of practice. And I feel that the colleges have met this responsibility well. Practically all of them hold annual conferences, postgraduate courses, so to speak, for the reëducation of the men in the field.

EDUCATION PRAISED

Our profession has grown to its present proportions largely through its flair for education. We should never overlook the fact that the first great impetus of the profession came as a result of the struggle of the A.V.M.A. for veterinary education, back in the middle years of the past century.

We are indebted to five veterinarians from Europe—Liautard, Smith, McEachran, Law and Detmers—each of whom founded a veterinary school, for the first organized veterinary education in North America. These pioneers and their pupils are largely responsible for the progress that has been made in veterinary education and service in this country.

When I tell you that I was born and raised on a farm in New England, where they still cherish a few traditions, you will understand and perhaps pardon the "Spirit of '76" in certain portions of my remarks.

I glory in the fact that my boyhood was spent in these surroundings and I still further rejoice in the fact that during the early days of my professional career I was privileged to associate with some of the pioneers and founders of our profession. In 1906-7-8 I served my apprenticeship under the tutelage of a man, a noble practitioner, George H. Berns of Brooklyn, who was filled with the highest traditional ideals of professional life. These contacts have no doubt left impressions which have remained dominant in my perspective.

Now, we all recognize that the education of young men is the means by which a profession perpetuates itself. But it is just as important that we older fellows keep educated. And so I am glad to note that the many state association meetings, such as yours here today, are placing emphasis upon veterinary reëducation. I am certain that every veterinarian who attends such meetings as this goes home with a broader knowledge than he had when he came.

While I am on the subject of veterinary education, I should like to discuss one other phase of it briefly. Doubtless you are all aware that one year of college work or premedical training is a requirement for entrance to all colleges approved by the Association. In 1940, I understand, one of the colleges in the East will require two years of college work as a preliminary to entrance.

There are differences of opinion on this subject, of course. Some think more practical farm experience should be required, another group contends that some form of internship should be added, while a third group believes that a full college course is essential. In all probability, all three plans would do no harm. It certainly wouldn't hurt a college graduate to know how to milk a cow, hitch up a team of horses, clean a stable or hoe a field of corn, for he can not tell someone else how to do it if he does not know how himself. Fundamental knowledge in practical agriculture is a valuable asset to the successful practice of veterinary medicine.

INTERNSHIP RECOMMENDED AS VALUABLE ADJUNCT TO VETERINARY CURRICULUM

A properly supervised system of internship would undoubtedly be a valuable addition to the present requirements in veterinary education. Its proper supervision and application, however, present problems that are not easily solved. In any event, these suggested added requirements are worthy of careful consideration by those who guide the destinies of our leading educational institutions.

As I see it, we will not reach the goal of having the most nearly perfect veterinary

educational standards until all veterinary colleges require a seven-year course for a college degree either in agriculture, science or arts and a professional degree. Seven years may seem a long time to spend in learning to become a veterinarian. But I assure you from personal experience that it is not too long. Young men at the age of 16 or 17 have plenty of time ahead for training and development. Veterinary science, as you well know, is deep and exacting. If the graduates of our veterinary schools are to be capable of carrying on the scientific achievements of today, and of the future, they will need thorough training.

The establishment of such standards for education would surely constitute progress which could not help but be reflected in the future scientific standing of the veterinary profession.

As you men know, there are many problems before the national association and the profession. I would like to discuss a few of them with you.

POULTRY PRACTICE DESERVES GREATER ATTENTION FROM VETERINARIANS

We are all aware of the great strides which have been made recently in the treatment of horses, cows, hogs and small animals. Too few veterinarians, however, are aware of the opportunities which lie in the field of poultry practice, yet there has been great progress made in this field. In the eyes of the poultry industry, the veterinary profession of this country stands today at the crossroads in the control of poultry diseases.

This situation presents a serious challenge which we can not treat lightly. If the general practitioner does not help to control and prevent the diseases which threaten the billion-dollar poultry industry, then we shall see more and more of what we call "quackery." The poultryman needs and deserves help and will accept it from either the nearest or most reliable source available. The veterinarian is often overlooked. Furthermore, many farmers believe that veterinarians are not interested in poultry diseases or are incapable of furnishing intelligent service.

But more serious to our profession than being overlooked is the veterinarian's being called and giving a superficial diagnosis. When a farmer calls a veterinarian to attend a horse or a cow, we do not question the farmer's right to expect proper veterinary service. Poultrymen feel they have a right to the same quality of service.

Last summer the A.V.M.A. was proud to have one of the eleven days of the Seventh World's Poultry Congress named in its honor. It was emphasized at the Congress that many commercial poultrymen are using a few biological products, a limited number of agents for the control of parasites, and a small group of disinfectants in their disease-control programs. The average farmer from Pennsylvania to the Rocky Mountains, with his flock of 100 to 300 chickens, is still groping in the dark, depending largely upon a $2.00 bottle of patent medicine to control the diseases of his poultry.

It is within this great area that nearly 5,000,000 farmers own about 90 per cent of the total poultry population of the United States. The average veterinarian in this great Midwest section is in general practice and it is here that our profession needs to consider carefully whether or not we shall remain static, which will result actually in going backwards, or take definite steps better to safeguard the health of this industry.

In the selection of the special Committee on Poultry Diseases this year, I have endeavored to consider all factors—the practitioner, the field of research and an equitable geographical distribution of membership. The chairman, Cliff Carpenter, had, I believe, the first private practice devoted exclusively to poultry. Another member of the Committee, Dr. Brandner, is recognized as one of the outstanding poultry practitioners in the United States today. This committee is preparing a comprehensive program to stimulate a greater realization that if we fail in the next decade to accept fully our responsibility to the poultryman, then we must be prepared to witness poultry diseases controlled by those we today call "quacks."

I have requested this committee to present a full and comprehensive report at the Washington meeting this summer. It is a sincere hope of mine that every veterinarian who hears or reads the report will show by his attitude and activities that he fully accepts his responsibility toward this important branch of agriculture.

A.V.M.A.-A.A.H.A. PROGRAM EXPLAINED

As you know, the important matter of dog foods came in for detailed discussion at Memphis. The A.V.M.A. voted to coöperate with the American Animal Hospital Association in the latter's program of approving foods of recognized nutritional value and issuing a seal of approval.

The committee on foods of the A.A.H.A. has given much time and careful thought during the past four years to the building of the present program. The best interests of the manufacturer, the consumer and the veterinary profession have been considered. Men of experience in various fields have been consulted frequently. Excellent legal talent representing the manufacturer and the animal hospital association have coöperated in preparing the present contract of agreement between that association and the manufacturer.

The undesirable situation that now exists in the dog-food industry is obvious. If there are selfish interests within our profession, responsible for opposition and misinformation, they should be eliminated from consideration for the best interests of organized veterinary medicine. During the past 20 years the small animal branch of our profession has made commendable strides in the fields of surgery, diagnosis, medication, hospitalization and disease control, yet on the whole we are seriously deficient in the progress which we have made in the field of canine nutrition. It seems essential, therefore, that the A.V.M.A. participate in this work.

There have been some inquiries from members of the veterinary profession regarding the fee that is charged for the testing service. It has been pointed out that the American Medical Association does not make any charge for the approval of a food by its council on foods. The two projects, in a sense, are quite different. The American Medical Association, through its council on foods, does not undertake to approve a complete diet for a growing child, or even for the maintenance of an adult. The A.M.A. council on foods may approve an incomplete food, such as a special milk preparation, nuts or pineapples. The examination of such articles of diet presents a problem different from the testing and approving of a complete dog food. However, in a comparable situation, where the medical profession, through the American Association of Medical Milk Commissions, an integral part of the A.M.A., approves a complete food, certified milk, the producer, who in this case is comparable with the manufacturer, pays the bill.

Inasmuch as neither association (A.A.H.A., A.V.M.A.) has the laboratory facilities, the equipment or the personnel of a scientific staff capable of conducting the exhaustive tests prescribed for approval, or the necessary funds to meet the expenditures necessary for carrying on the work, it is imperative that the manufacturer in this case as well, for the present at least, must pay the bill. No one derives any monetary benefits from the program; the committee on foods and the advisory council serve without remuneration. The fees are used to defray actual assay, food purchases, clerical, and essential administration expenses.

It is possible for the scientific advisory council, in certain instances, as in some cases of A.M.A. approval, to accept protocols of tests made by a manufacturer or a broker of a given food. It is likewise possible to accept protocols of assays of a food for review, but it is felt that it would be undesirable to issue a seal of approval on the basis of such protocols alone, as this would place on the associations the responsibility of classifying laboratories, in addition to testing dog foods.

In the case of a food tested and found inadequate, provision has been made for the manufacturer to improve his product and resubmit it, should he so desire. If he is unwilling to do this, the application

for the seal of approval is rejected and the profession and the public are advised accordingly. Practically an identical situation exists in the control of production and the approval (by seal) of certified milk by the A.A.M.M.C.

The A.V.M.A. is not entering blindly into a coöperative experimental venture. Its executive board made a thorough investigation of the methods of administration, the purposes, the agreements, and the contracts and the liabilities of the program before the resolutions were referred to the House of Representatives for adoption. In asking the veterinary profession as a whole to participate in this work, the executives of the Association are doing so with the belief that it is for the best interests of the profession at large and of organized veterinary medicine. This is not a program which can be carried on successfully by a committee. It requires the coöperation of all veterinarians, particularly those who are practitioners. As practitioners, we should acquaint pet owners with the text of our official seal and the ideals for which it stands.

Every veterinarian should familiarize himself with the fundamentals of this work. Many dog-food advertisements, as well as radio script, continually quote veterinarians. The inference usually contained in such copy is in effect that the veterinary profession endorses that particular product or claim. Actually, this is not the case. The phrases "veterinarian" and "veterinary profession" are being used too freely. We are being misquoted as a group because some individual veterinarian has expressed a personal opinion on canine nutrition. Steps should be taken to discourage such a procedure. The above program, as outlined, constitutes the official opinion of organized veterinary medicine. All veterinarians, therefore, should insist that companies wishing to quote us comply with the regulations which we have adopted. Veterinarians are frequently approached by manufacturers of dog food for information either about this program or some phase of dog nutrition. The committee on foods of the A.A.H.A. will gladly assist you, if you wish, in handling these inquiries.

I am sure that, with the aid of the forward-looking veterinarians throughout the country, the A.V.M.A. and the small animal branch of the veterinary profession can carry on this progressive work.

ASSOCIATION'S PUBLICITY PROGRAM IS PROMOTING WELFARE OF ENTIRE PROFESSION

During the past two conventions of the A.V.M.A.—in New York and in Memphis—we have had extensive publicity of the activities of the Association. This publicity has been secured not only in newspapers and magazines throughout the country but over the air as well. During the Diamond Jubilee convention in New York there were seven radio talks, and during the Memphis convention there were twelve, all made by leaders in our profession.

In addition, there has been a great deal of publicity throughout the past year—both in print and on the air.

The publicity campaign carried on by the Associated Serum Producers in the Central West is worthy of special mention. No one can give an accurate estimate of its far-reaching results to the profession at large and to the practitioner in particular. Any and all publicity that has the objective of educating the livestock and animal owners as to the value of qualified veterinary service is of great value to the profession.

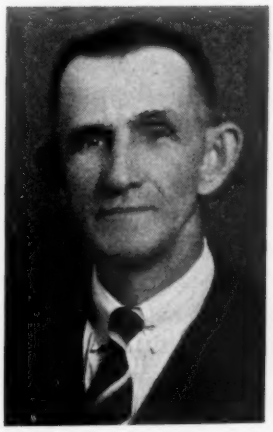
This publicity is the result of a deliberate campaign by the A.V.M.A. and many allied interests to inform the public about the services of qualified veterinarians. It is a campaign which will be continued throughout 1940. The officers of the A.V.M.A. believe that the campaign is of great value to the veterinary service. To be a complete success, however, the campaign needs the enthusiastic coöperation of every man in the profession.

Here is what one prominent veterinarian in the Central West has to say regarding this publicity and public relations program:

> Believe it or not, the publicity the A.V.M.A. is giving to veterinary service is

(Continued on page 384)

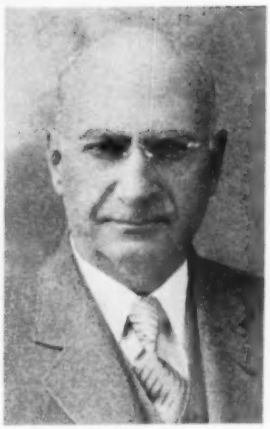
Here Is Your House of Representatives

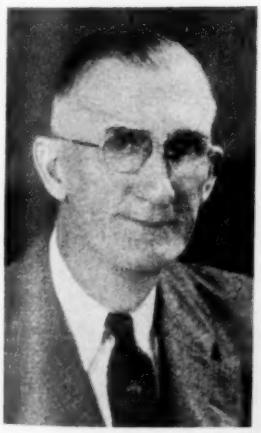


Top: I. S. McAdory (left), Alabama; T. B. Jones (right), Arizona. Center (left to right): C. D Stubbs, Arkansas; G. D. Pinder, British Columbia; H. S. Cameron, California; Thos. E. Traylor, Colorado. Bottom row (left to right): F. F. Bushnell, Connecticut; R. M. Sarde, Delaware; Mason Weadon, District of Columbia; J. L. Hopping, Georgia.

Below: F. M. Wilson (left), Iowa; E. J. Frick (right), Kansas.

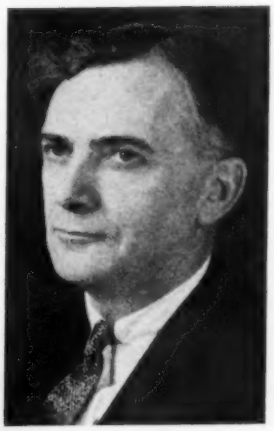
With but four exceptions (J. H. Yarborough, Florida; J. F. Witter, Maine; R. H. Lay, Manitoba; and M. B. Starnes, Texas), the portraits of the delegates to the House of Representatives, as of February 1, 1940, are reproduced herewith. Aside from a few changes which will take place through elections at various state and provincial association meetings, this is the delegation which will represent the interests of the membership at large during the 77th convention at Washington, D. C., August 26 to 30, 1940.





Above (top to bottom): A. K. Kuttler, Idaho; D. E. Sisk, Illinois; J. L. Axby, Indiana.

Below: D. E. Westmorland (left), Kentucky; J. D. Jones (right), Louisiana.



Above (top to bottom): L. J. Poelma, Maryland; L. A. Paquin, Massachusetts; B. J. Killham, Michigan.

Top (left to right): L. E. Stanton, Minnesota; R. H. Stewart, Mississippi; A. T. Kinsley, Missouri; H. F. Wilkins, Montana. Bottom (left to right): Floyd Perrin, Nebraska; Wm. R. Smith, Nevada; C. L. Martin, New Hampshire; R. A. Hendershott, New Jersey.

(Continued from page 381)

receiving nationwide attention. Our bold statement that without a well-disciplined veterinary service our country is doomed, is beginning to make folks take notice and, if continued consistently, this new analysis of what our service means to the people will soon have a telling effect in the status and development of our undertaking.

We in the Middlewest are beginning to notice a remarkable change in the attitude of those who were once hostile to us. The change is marked, unbelievable. Agricultural leaders are opening their eyes to the fact that "agriculture is animals" and "animals these days require better veterinary service." When all the facts become generally known, our destiny is set for all times.

My subject, as announced, is, "What the Practitioner Can Best Do for the A.V.M.A," and perhaps it is time that I began to discuss this. If I may, I would like to amend this title somewhat to read, "What the Practitioner Can Best Do for Himself." I am convinced that in serving his own interests, the practitioner also will serve the interests of the national association.

Of course, I have already indicated certain things which the practitioner can do to benefit himself. These are: To keep abreast of new developments in the profession, to accept responsibility toward the field of poultry practice, to coöperate in the Association's rabies program, and to aid in every way possible the dog-food-testing program. There are other things, however, which the practitioner can do that will not

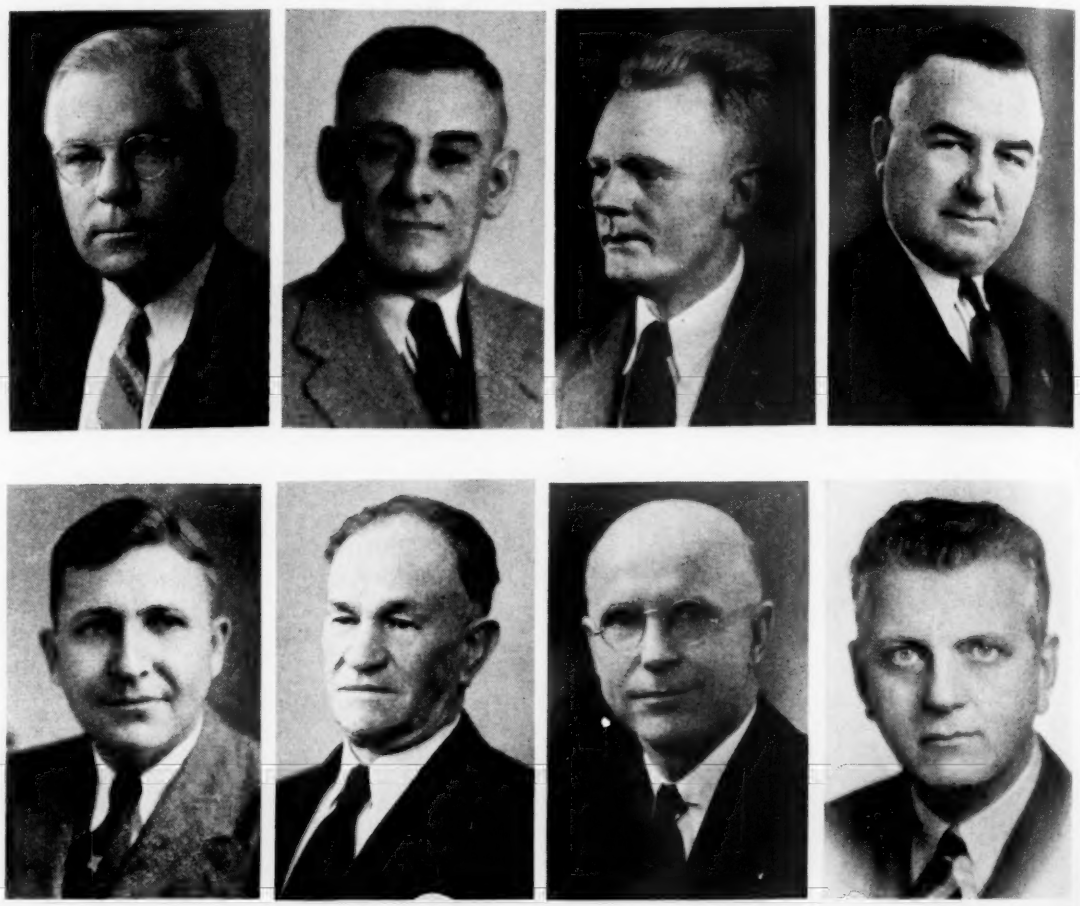
only benefit him but will materially help the Association.

First and foremost, I put the need to join the Association. In every sense of the word, it is your association. Those of us who are at present charged with responsibility desire to do all we can to acquaint you with the aims and policies of the A.V.M.A. We shall endeavor to present from time to time, in advance of the annual meetings, items of importance that will come up for consideration. The publication of the proposed constitution, administrative by-laws and code of ethics well in advance of the meeting, in order to permit discussion and consideration at various state meetings, exemplifies this thought.

SCOPE OF ASSOCIATION'S ACTIVITIES IN RELATION TO FINANCES EXPLAINED

Beginning with the January 1940 issue of the JOURNAL is a new section, entitled "A.V.M.A. Activities." This was inaugurated in order to keep the membership posted on important items of interest, actions of the Executive Board, the Board of Governors, etc. For instance, in February you probably read the item that the Executive Board recommends an increase of $2.50 in the annual dues. In view of a larger and better journal, an increased publicity-public relations program, increased personnel and service in the central office, contact with component associations, etc., we are giving out more than we are taking in. This can not go on very long. If this program is to

Top (left to right): F. L. Schneider, New Mexico; R. R. Birch, New York; A. A. Husman, North Carolina; T. O. Brandenburg, North Dakota. Bottom (left to right): Reuben Hilty, Ohio; H. Wood Ayers, Oklahoma; H. R. Potter, Ontario; O. H. Muth, Oregon.



Top (left to right): Wm. H. Ivens, Philadelphia; J. S. Barber, Rhode Island; W. A. Barnette, South Carolina; Geo. E. Melody, South Dakota. Bottom (left to right): John H. Gillmann, Tennessee; N. C. Spalding, Utah; G. N. Welch, Vermont; I. D. Wilson, Virginia.

be continued, if the membership want it continued, we must increase our revenue, we must balance our budget.

During the past three years the reorganization program has been expensive. There have been a few unusual expenses. The outlay of approximately $5,000.00 for the exhibit at the New York World's Fair was one that will not need to be repeated. However, many of the millions of people who visited the medical and public health building, where the A.V.M.A. exhibit was located, must have formed a favorable impression regarding our profession. This year the government demanded payment of $1,000 in principal and fines for Social Security. We had been advised by counsel that the Association, being a non-profit professional organization, is exempt; however, this supposition proved to be erroneous. Unusual and unforeseen local expenses connected with meetings developed during the year. The greatly increased size of the JOURNAL from 74 readers pages in January 1939 to 164 reader pages in February 1940, or an increase of 90 pages in the reader section, has added greatly to the expenses. It costs almost as much per year to deliver the present journal to each member as he pays in dues; the publicity-public relations program this year involves a comparable expenditure per member. Therefore, at this rate, each member is receiving, in these two items alone, almost twice

as much as he pays for and, in addition, he derives all of the benefits of membership in our national association. Certain economies are planned and certain expenses encountered the past year are finished. There has been a substantial increase in membership; in fact, 1939 was a record year. However, in order to maintain a satisfactory working balance, it is imperative that the Association have an increased revenue.

If we are to maintain the progress already made, it will cost each of us a little more in dues. It will be easy to go back to the old order of things, it will make less work for everyone. It is up to the membership to decide. I personally hope they will uphold the action of the Executive Board in this item. In an endeavor to make these things possible, workable and permanent, the executive officers solicit not only your membership and financial support but your constructive suggestions, your criticisms and your coöperation.

Our three distinguished and immediate past-presidents, Foster, Brumley and Bergman, the chairman of the Executive Board, Harry W. Jakeman, and the reorganizing committee, have worked hard and long for a better administrative set-up. They have set a fast pace for those who follow them. They have accomplished much. The official journal, the central office, the executive branch and, I am sure, the rank and file of the profession are all pulling on the front end of the load for a bigger and better national association. Our goal is, "Every qualified, ethical veterinarian in North America, a member." With your help, we shall attain it. In saying it is your association, I mean just that.

STATE ASSOCIATIONS SHOULD SELECT PRACTITIONERS FOR HOUSE OF REPRESENTATIVES

Secondly, there is the matter of delegates to the annual meetings of the Association. If the members of each state association will send the best qualified, most successful practitioner in their state to the A.V.M.A. convention as their delegate to the House of Representatives, and pay his expenses, there will be no question






Top: E. E. Wegner (left), Washington; S. E. Hershey (right), West Virginia. Below (left to right): J. S. Healy, Wisconsin; L. H. Scrivner, Wyoming; H. M. O'Rear, National Association of Bureau of Animal Industry Veterinarians; R. A. Kelser, Veterinary Corps, United States Army.

about the future of your national association. As closely as we can estimate, 60 per cent of the members of the A.V.M.A. are practitioners. With this majority, if the practitioners let control get out of their hands, it is their own fault.

If practitioners complacently send representatives of educational, research, regulatory or commercial groups to the conventions as their delegates, and if the votes of these delegates are influenced by their personal ideas and training in a way that may be at variance with the best interests of the practitioners, then I say as a practitioner that we have no one to blame but ourselves. Send practitioners to the annual conventions as your representatives and you will have the representation you should have.

PRACTITIONERS URGED TO CONTRIBUTE ARTICLES FOR JOURNAL

There are two other matters in this general subject of being helpful to the A.V.M.A. One is to send interesting and unusual case reports to the JOURNAL. Similarly, may I urge you to send in new and practical suggestions in medical treatment, surgery, feeding and the handling and restraint of animals. In this way you will be contributing splendidly to the sum total of the knowledge of the profession.

Last year the Association took into membership between 400 and 500 graduates of the class of 1939 without requiring the payment of the usual initiation fee of $5.00. They paid their portion of the annual dues, as is customary, the idea being to encourage their membership and not only give them a little boost financially when they were entering upon their chosen professional career, but also to give the Association the benefit of an infusion of new blood, men with the very latest ideas and training. This policy will be continued and, undoubtedly, you realize what this will mean to the Association in 20 years. It will mean a membership of approximately 12,000, or nearly 100 per cent of the profession, of which 7,500 to 8,000 should be practitioners.

As I see it, the practitioners as a group are so busy making money, or attempting to make money, that they will not give the time necessary to serve their national organization and, in turn, serve themselves. They want to "let George do it." I don't know that I blame them.

During the past 20 years we have had about 25 to 30 per cent practitioners as presidents of our national organization. I think that everyone of them deeply appreciated the honor but I also think that, before the year was over, they realized a substantial decrease in revenue, for, after all, about the only thing a practitioner has to sell is service. There are only so many days in the year and every day he gives for the "good of cause," there are just so many days less that he can give for the good of himself. Personally, I realize this keenly.

We, as practitioners, have it within our power to have a practitioner as president of the A.V.M.A. every year, we have it within our power to have 100 per cent representation on the Executive Board, we have it within our power to have 100 per cent practitioner membership in the House of Representatives. But, just as soon as we divorce ourselves from the influence and benefits of our educational institutions, from the benefits of the service we demand, and get, these days from the commercial field, from the support, assistance and help that we receive from those engaged in regulatory work, we will begin to witness the beginning of the end. To be sure, such a situation will not occur. However, I personally hope that this year we may witness a still more unified profession and that those of us who may be classified as practitioners may more fully realize our duty to ourselves to better serve our national organization. We get out of it just what we put into it—no more, no less. It is the duty of each member to do his share.

And while I am on this subject, I should like to comment on a trend which I personally regard as unfortunate, that of going off on tangents and forming side associations. When veterinarians do this, they merely weaken their national association and weaken their individual status. The members of the profession as a whole should support the national association they already have. Surely, in unity there is

strength. I can assure you that the A.V.M.A. is not controlled by any clique.

Throughout the world the whole philosophy of individual liberty is under attack. In the history of the United States we find that freedom has released man's creative genius and initiative, while under European dictatorships people have willingly surrendered every liberty to some man or group of men who have promised economic security, moral regeneration, discipline and hope.

The United States is at present free from these stultifying influences, and I trust that we always will be. This new year, perhaps more than ever before, we should be thankful that we live in such a country. The bulwark of freedom lies in the independence of the individual. Let us all strive constantly to maintain that independence, that freedom, which is our heritage.

The A.V.M.A. was organized during the throes and depression of a great civil war. The profession has gone on from decade to decade toward achievements little dreamed of in yesteryears. As Walt Whitman says in the "Song of the Open Road," "It is provided in the essence of things that from any fruition of success, no matter what, shall come forth something to make a greater struggle necessary."

With the increasing demand by animal owners for better, more efficient, more scientific veterinary service, with a keener desire on the part of the practitioner to render such service, with the rising standards in education and with still greater accomplishments in research, there is no question in my mind about the successful future of the veterinary profession.

Ajax

Ajax lives in history as a symbol of audacity and folly—as a brave soldier who didn't make good. This mightiest of warriors defied the lightning as well as the enemy. But he came to a bad end. He suffered moral defeat and killed himself because Ulysses, more prudent and wise, won the armor of Achilles by coöperation and sagacity.

In striving to effect a more solid organization of the veterinary profession one is often reminded of this tale of Homer. The veterinarian fighting single-handed in his community is a veritable Ajax—defying the lightning in vain.

A recent release of the U. S. Department of Agriculture says that the agricultural agents (of both sexes) have organized and have a working committee in every county, constituting a network of more than half a million voluntary leaders. Inasmuch as the motives of this gigantic organization are of the highest order, guidance from an organized veterinary service in every community is a means to an end that no fighting Ajax can hope to achieve.

Dual Duties

All agree that the use of local veterinarians in the public service—municipal, state, federal—is the current trend. The status of them in the dual rôle is, however, a detail to be worked out. For example, the compensation is not always attractive, especially to the busy practitioner, who sometimes sacrifices more than he receives. The price paid for veterinary service is sometimes entirely too low to be inviting, and the same also may be said of full-time jobs in the state and federal service. The fact that the qualifying requirements are as high and even higher than those of other professions remains to be told, but can be told and acted upon only when veterinarians in and out of these offices realize that the national and state organizations are the only instruments available for improving their rating, their pay and their work.

Comparison of conditions today with those at the turn of the century or even 20 years ago should, however, convince the most skeptical that the veterinary profession is climbing. For the first time the people are becoming aware of its relations to popular welfare. The governing verve is the wise leadership of the state and national associations which, thankfully, are not easy to sway from the path of righteousness and wisdom.

EDITORIAL

Every addition to knowledge is an addition to human power.—Horace Mann.

Historic Rinderpest

RINDERPEST, or cattle plague, is little known to the American veterinarian. Except for those who served in the Philippines it is but the name of a grave disease of cattle prevalent in oriental countries, regardless of the fact that it is not, to any important degree, a respecter of geography. Where cattle can exist, so can rinderpest. We know it as an oriental disease because the competent veterinary services extant in occidental countries have driven it out and, by eternal vigilance, keep it out. We once heard Chief Mohler of the federal service remark that rinderpest is one of the constant threats to American cattle which the United States bureau of animal industry is prepared to handle should the disease by chance crash the gates of our guarded international boundaries.

The potentiality of this cattle plague is nothing less than a threat to our civilization. Were it to get a foothold in the congested cattle population of the United States through peacetime incompetence of the veterinary service or through military necessity, rinderpest would be a more powerful enemy than all of the armies and navies of the world. The reader is referred to history for the details of the sweeping epizoötics which raided continental Europe before systematic veterinary protection was established or when effective protection was overwhelmed by wars. The disease has followed in the wake of armies in all wars of history, including the World War. The last outbreak in continental Europe occurred in 1920 in Belgium, where it raged from June to October before it was stamped out, at tremendous cost not only to Belgium but also to adjacent countries (including our own), which found it necessary to maintain restrictions against the importation of carrier mediums.

The record of the Belgium outbreak is revealing. It started from a shipment of zebus from India unloaded at Antwerp to be reëmbarked for South America. Several of them died and the rest went on to Lisbon and Rio de Janeiro. Notwithstanding the source of these animals, no autopsies were held and no diagnosis was made. Rinderpest was not suspected, not even after three shiploads of cattle from Baltimore and New York, herded in the same locale and shipped by rail to Belgium abattoirs several weeks later, carried the infection to their various destinations. When these animals came in contact with some German cattle destined to farming districts, the disease was scattered far and wide—to 40 different foci. It was not until the disease appeared in these rural districts and was studied by the Pasteur Institute of Brussels and the veterinary school of Cureghem that the trouble was identified as rinderpest.

The fact that numerous sick animals were slaughtered *d'urgence* and others died shows the importance of meat inspectors' keeping better informed on the pathological picture of rare but important diseases. Until positively recognized, the disease had been passed over as an atypical form of foot-and-mouth disease (*Revue générale de Médecine vétérinaire*, November 15, 1920, p. 577). The self-isolated, non-reading practitioner as well as the self-satisfied meat inspector, serenely contented with their knowledge of current matters, are dangerous men. This is a none too gentle reminder of the responsibilities assumed by those engaged in the application of any branch of veterinary medicine.

The world is now at war. Animal diseases will follow in the trail of the armies. The veterinary services will be taxed to the limit of their capacity. Military necessity is emptying the medical and veterinary schools, physicians and veterinarians are at the front, rural districts are left at the mercy of the aged and infirm professionals, and the vigilance of veterinary services which insure an amplitude of food in peace times is curtailed. *C'est la guerre*, today as in all times. Though mankind is slightly more aware today of his dependence upon domestic animals than in times gone by, the practice of veterinary medicine in the minds of the average American is still an unknown or insignificant pursuit. Its place in peace is scarcely realized and its responsibility in war is an unstudied chapter of our social set-up. The industrialists deplore the aid farmers receive and the farmer, sometimes unwisely coached by his organizations, is little aware that the doctors of his animals and their contemporaries in the public service are the salvation of his enterprise.

All of this the American Veterinary Medical Association, with its background of time and achievement, is delegated to iron out and keep in order. It is the instrument of the veterinarian and, through him, of the people, to be used to prevent tragedies which only the most studious historians ever have estimated—the tragedies arising from uncontrolled animal diseases.

"Scientific" and "Practical"

IN THIS issue a new column, entitled "With the Editors," makes its bow. Its main purpose is to determine to our own satisfaction, and if possible to yours, whether the JOURNAL is or is not a "practical" magazine. We want to find out whether the white space filled up with so-called ultrascientific material contains anything that can be put to work in the every day life of a veterinarian, contrary to a too general impression (we think) that much printers' ink is wasted.

We would regret to see our former colleagues with whom we have labored for many years in the clinical field abandon the science that begets their art. We believe it would be unfortunate for the practitioner to fade into the land of the skillful but unscientific artisan, because science not only creates but underwrites everything the clinician does. The so-called scientific article of today may and often is the practical one of tomorrow. For this we thank the demesne of research and investigation, where work goes on continuously to keep the practice of medicine within the bournes of respectable occupations.

No one can gainsay nowadays that medical science has not marched far ahead of its use. The reason for this, at least in part, is the neglect of practitioners to seize its teachings and put them to work without waiting for a calamity to come along as a reminder of forgotten facts. Illustrative is the article in the December 1939 issue by Meyer, Anderson and Eddie on Weil's disease in the United States, occupying 18 pages of graphs, tables and running material describing in detail an entity once thought to exist only in certain countries of Europe but which, the authors found, is widespread throughout this country. No salient phase of the disease is not described. Is this article "scientific" or "practical?" When even the children know Weil's disease, it will be practical. Today, because the hunger for knowledge on such uncommon matters is not great and time too short, the article is "too scientific."

This example applies equally well to all of the articles published in the body of the JOURNAL in recent months. It is an example for practitioners to ponder. As a rule, articles of the class in question are impractical only to those who do not trouble to analyze the author's theme. This we shall attempt to do for our readers in the new column, "With the Editors." Why? For one thing, we know (from experience) that time is a factor to be considered in a practitioner's daily grind. But, this does not imply that the careful reading of scientific articles should not be encouraged. A medical education is never finished, and the I.Q. of its members establishes the rating of a profession. So, don't keep the rating low.

Who Is a Practitioner?

THE TEACHER, pathologist, sanitarian and food inspector are all practitioners, provided that they work at their specialties. Custom, however, wills that the word be reserved for those who treat the sick in a private capacity as an occupation. With this in mind in our *métier*, an inquisitive correspondent wants to know how much public service a veterinarian is permitted to do without losing his right to the name *practitioner* or, if engaged primarily in the public service, at what stage does he sacrifice his sworn allegiance when permitted to practice privately as a sideline.

These are questions to ponder. They come from a member who writes further: "When is a practitioner not a practitioner?" The inquisitor thus complicates matters by asking where to classify those who divide their time between public and private work and, furthermore, wants to know why a public official is not as much a practitioner as the one who does the same work for fees collected from the owners. It seems that this colleague picks up samples of milk and works in the local abattoir in the morning, spends many afternoons inspecting dairy farms, and treats the sick animals of the community between times, which is the ultimate desideratum.

These questions connote insight and foresight, since the general trend of the veterinary service is toward using the local veterinarian in the public service on an ever-increasing scale. The object, so clearly stated by the writer of the letter, is the building of a better veterinary service for the American people at the lowest possible cost. But, what too many overlook is that the Association has been working to that end since the middle of the nineteenth century—since 1863.

The Association's achievements are safely posted in the minds of the thoughtful. Starting from absolutely nothing, it promoted the making of a bureau of animal industry and the system of state and assistant state veterinarians, of a veterinary corps for the army, of the federally accredited practitioners, of the drafting of local practitioners for disease-control drives, of allocating public work to private hands, of founding local meat-inspection programs and, all the while, governing the development of an educational system to furnish a qualified personnel to carry out these projects. A reminder that all this was Association-made is boastful but true. The biography of the Association and its officers and members is the evidence. The beneficiaries have been the nation, the veterinary profession and the veterinarians themselves. The progress made corresponds to the amount of coöperation these men were able to mobilize from the latter and the amount of opposition from them they were able to overcome.

Wisdom was never a universal virtue of any group of human beings. Veterinarians are not exceptions. In the councils of the Association the building of a good name for veterinarians in all branches was kept uppermost in mind. Money-making was judiciously tabooed, though not always without considerable criticism. Every period has seen money-making crop up at the expense of the altruistic motives. He who will but glance through the Association's archives is soon convinced that there was no moment in the past 77 years that the national association was not upbraided for not doing the wrong thing, for doing things that society and science might disapprove and time discard. Speaking in understandable terms, it was never easy to keep the practice of veterinary medicine from sinking to a pretty low level.

At every step, the absence of selfish motives had to be proved and re-proved to convince a skeptical public and their legislators. Scientific control of tick fever, tuberculosis and hog cholera (as examples) was denounced as a design of money-making veterinarians until the wisdom of the projects became apparent. The word racket had not been coined. Brucellosis and rabies are now running the same gauntlet. But for wise discretion it would now be easy to make a false step difficult to live

down. But, no false step will be taken. Tradition forbids.

The veterinarians of the United States are not thoughtless children. The majority are conscious of being well advised and the reward for their obedience is an unblemished profession that has risen from obscurity to a place of esteem in the economic structure and the hearts of the people. Money can't buy such a heritage. Let us look at the forest as well as the trees. We are all practitioners of an art mapped out by an important branch of science, regardless of how the family budget is collected, and let's not forget that the moment "the good of the people" is erased from our banner, we are on the way down.

Defining "Antiseptic"

CONFUSING definitions in medical dictionaries as well as in dictionaries of the general language have necessitated the drawing of a line of demarcation between what is and what is not antiseptic. Fair and impartial enforcement of laws governing the labeling of medicinal products of that class requires that such a line be drawn. A glean through dictionaries shows but little accord in defining the word. The word *putrefaction* and the etymon *sepsis* are used synonymously regardless of the fact that the one is but the end process of the other. Living matter can putrefy only after it has been reduced to a non-living state, yet in defining the word *antiseptic,*

a) *Webster's* says: "*adj.* Tending to prevent or arrest putrefaction. *n.* That which may be used to destroy bacteria with little or no harmful effect on the living body."

b) The *New Century Dictionary* says: "*adj.* Pertaining to or effecting the exclusion or destruction of the microörganisms that produce sepsis." [*Sepsis is defined as putrefaction.*]

c) Sollmann says: "*n.* Substances that merely suspend the viability of bacteria are called *antiseptic;* while those which kill them are *germicides.*"

d) *Larousse Médicale* says: "*n.* Medication against putrefaction, that is to say, that which acts by destroying harmful microbes."

e) *Stedman's* says: "*n.* A substance which prevents the action of the germs of fermentation, decomposition and disease."

f) The Food and Drug Administration (U.S.D.A.): "*n.* The term applied to an article intended to be used in such a way that it remains in contact with the tissues for but a brief period of time connotes *germicides.* On the other hand, the term may be properly applied to an article that possesses only inhibitory properties if the directions for its use require long contact with the body."

While *sepsis* and *putrefaction,* the one from Greek and the other from Latin, mean rotten, the derivations have not been sustained by usage.

Book Notices or Reviews

A REVIEW of a book, entitled *Biological Products,* printed elsewhere in this issue, and of another, entitled *Poultry Practice,* bring to mind the advisability of drawing attention to the Book Notice section of the JOURNAL. As a rule, these articles contain useful information on matters of current interest and also serve as a guide for the prospective purchaser.

Interviews with readers of the JOURNAL here and there show that book notices are by no means universally read, notwithstanding the hours of study each one of them represents. To promote the reading of these comments on new literature and to encourage the writing and reading of good books, we have departed from the conventional style to the extent of adding illustrations from the text to these articles when suitable ones can be obtained. For this departure we apologize to the book worm who seizes ravenously every new addition to the literature with the view of improving his technical education and keeping abreast with the times. As pointed out before, many of us have lost our thirst for good reading material and reference books while some of our clients, edified with the lauwine of bulletins on veterinary medicine and hygiene coming to their homes, may have stepped ahead of us in literary attainments. In short, they may know more about new veterinary literature than we.

We name the column "Book Notices" rather than "book reviews" because we do not always pretend to write a true image of the authors' thoughts. But we do attempt to tell readers what the book contains and how it impresses us.

The Sketchy Control of Rabies

THOUGH vaccination of dogs against rabies has brought out many conflicting opinions, no one has questioned the virtue of ridding the country of the straying dog. The handling of strays stands first on the list of control measures and will continue to stand first until the virtue of vaccination becomes less vulnerable.

Granted that killed rabies virus has sufficient immunizing value to aid in the control of the disease, there are other scientific facts that the clinician must face. The concentration of the virus in the brain tissue suspended in the vaccine is said to lack the uniformity of a perfect immunizing product. Some samples taken from the open market have been found to be practically inert by unbiased pathologists. Others were found to have oscillating values. These are facts the clinician must face to avoid criticism. Going off on a tangent because vaccination is challenged on scientific grounds is not good defensive strategy. Moreover, until the disposal of stray dogs is kept foremost among control measures, the exponents of compulsory, universal vaccination of dogs against rabies are apt to be blamed for having only selfish desires.

The Committee on Rabies of the United States Live Stock Sanitary Association for 1938 recommended that the control of rabies be placed in the hands of the federal government in coöperation with the states and that suitable laws be passed and ample appropriations made to carry out a sustained control program. The Committee was composed of H. C. Rinehart, chief veterinarian of Illinois, *chairman;* H. W. Schoening, U. S. bureau of animal industry; and M. F. Barnes, Pennsylvania bureau of animal industry. They pointed out the stray dog as the reservoir of the virus. It was stated that if stray dogs were destroyed, the eradication of rabies would be accomplished but that local efforts do not receive the support of any group of citizens, of the press, or even of the small animal practitioners because the attachment of man for this "animal of special privilege" blocks the best efforts of local enforcement agencies, despite the large monetary losses and anguish rabies continues to cause. During the year in question, 30 persons died of rabies in the United States, and the loss in farm animals and valuable dogs ran into high figures that never have been computed. The need of a nationwide control project was not questioned and, since then, the thought laid down by the Committee has crystallized inside and outside of the veterinary profession, with the prospect of meeting with the same universal approval as the gigantic campaigns which have brought other widespread contagions of domestic animals under control.

At the present time, the control of rabies *is* sketchy. Judging solely from the Association's correspondence, two trains of thought are dominant: The one would keep rabies control in the hands of local health departments and the other in the government's disease-fighting forces. The latter plan was approved at the Memphis session (1939).

"What will be the fate of vaccination when the government takes hold?" is a question asked. While the JOURNAL is not a spokesman for the government, the answer is easy. No useful measure is ever discarded in disease control.

Something for Minus Nothing

A THIEF who stole an automobile wrote to the owner for the certificate of title in order that he could collect for the sale of the car. . . . A boy murdered his father and mother and asked for mercy from the jury on the ground that he is an orphan . . . a veterinarian, down state, graduate of a dubious college, writes for protection against a practicing quack but does not belong to any local, state or national association.

The gasoline taxes for 1938 cost the people of the United States $766,853,000.

WITH THE EDITORS

AMONG SUBJECTS ranging from black widow spider bites in dogs to pneumonia in race horses, every reader should find at least one or two items of genuine interest in this issue.

The leading article, "A Historical Prospectus of the Washington Session," blends events of a meeting of the Association in Washington, D. C., 49 years ago with what is in store for those who plan to visit the national capital in August. The attendance, of course, will be larger than in 1891 and the committee in charge will sell banquet tickets for less. (Note on page 295 the price of $5.00 for a ticket to the banquet of 1891.)

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President Way's address, "What the Practitioner Can Best Do for the A.V.M.A.," is a stimulating delineation. The dog-food-testing program, publicity and public relations are discussed at length. Much is said concerning the JOURNAL.

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The article on "Listerella Infection in Swine" by Biester and Schwarte is of interest to the swine practitioner and research worker, since these authors report for the first time that infection in swine. Their description of the clinical picture is excellent, and their well-grounded experimental data place this important contribution upon tenable ground.

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Those who attended the Memphis session will remember W. A. Aitken's paper on "So-Called Hemorrhagic Septicemia." Aitken is an experienced Corn Belt practitioner. He points out that, at least in his practice, Pasteurella infections are rare. His findings are backed by bacteriological examinations conducted at a diagnostic laboratory. Aitken declares that "hemorrhagic septicemia" is but a handy name to tack to obscure disease processes. So, as this Iowa veterinarian says, "When a diagnosis of hemorrhagic septicemia is made, be sure to bring along an affidavit."

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Another convention paper, "Diseases of Feeder Cattle," by W. S. O'Neal, takes the practitioner into the feedlot for a short, direct and practical discussion. The popularity of this paper brought O'Neal to the Illinois state meeting in February.

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J. D. Jones of the University of Pennsylvania medical school gives the busy veterinarian pertinent information on vitamins without repetition of the known facts about their effects on laboratory animals.

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Proctor of Kentucky, whose practice is limited to equine medicine, especially to Thoroughbreds, discusses the modern conception of pneumonia in horses in a thorough manner.

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Norman R. Stoll of the Rockefeller Institute for Medical Research, Princeton, N. J., whose style of writing is refreshing, discusses immunity to helminths. Small numbers of worms received by the host over a relatively long period leads to a specific immunity. Usually, too many in a short period provoke clinical symptoms. Nutrition, chilling, breeding, lactation and physic influences play an important part in establishing and maintaining resistance. There is a time when an infestation becomes so great that the host is able with one surging effort to expel the parasites. "Scouring is a common manifestation of this reaction," the author says.

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Swanson, Harwood and Connelly report the successful use of phenothiazine for the removal of adult ascarids in swine and, of course, the nodular worms, too. Interesting is the fact that this chemical, which is a close relative of methylene blue, was dis-

covered 75 years ago and yet its true virtue remained unknown all these years.

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A formalized, lung-tissue vaccine for the control of mink distemper is reported by Henry Pinkerton of St. Louis, Mo. In addition to its preventive properties, the vaccine appears to have some curative value in the early stages of the disease. The vaccine is standardized by the number of inclusion bodies present in the tissue used in the production of the final product. Since there is considerable difference in the virulence of the strains of virus, the vaccine should be prepared from the specific strain responsible for the outbreak. Pinkerton is a bit inconsistent in mentioning the work of De Monbreun, since it is now believed that the latter was working with fox-encephalitis virus rather than distemper virus.

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"Anthrax in Minks" by Stiles and Davis points out the importance of competent veterinary inspection of fresh meat.

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Gassner and Thorp report something new on the sheep tapeworm, *Thysanosoma actinioides*. The report is a fine contribution by veterinarians to the science of helminthology.

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The use of silk sutures and a lesson in abdominal surgery are nicely handled by John D. Gadd of Maryland. Everyone who uses a suture needle will find useful information in this article.

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Lead-arsenate poisoning of sheep and cattle is discussed by McCulloch and Smith. The detailed history of cases and the clear description of the clinical tableau together with the feeding experiments give a comprehensive view of the subject.

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Metzger and Stokes employ the ever-fascinating chick embryo to propagate two strains of *Brucella abortus*. Even after eleven passages, these strains did not evidence any changes in virulence, cultural characteristics or antigenic properties.

Wayne H. Riser writes of a new tumor of dogs. It is the first report of its kind in the literature. Riser is a busy small animal practitioner who takes the time to improve his education by attending a leading midwestern college between office calls. We have asked Riser to prepare some case reports on functional disturbances in dogs for future issues of the JOURNAL.

Re: The February 1940 Issue

Graham, Hester and Henderson, University of Illinois, in reporting four outbreaks and confirming experimental work, showed (pp. 135-140) that blazing away at clay pigeons in pasture lots is not a harmless pastime. Shattered and whole (the ones you miss), the discs known in the field of sport as clay pigeons may be consumed by hogs with deadly effect. The coal-tar pitch they contain produces terrific damage to the liver and, inasmuch as coal-tar pitch is a component of other commodities around the farm, this observation and research adds another agent to the index of toxicology and tells precisely the damage it does. Thus, "flake after flake" the JOURNAL drops new utilitarian facts to the field of clinical veterinary medicine.

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Among the practical papers read at the Memphis session none is of more interest to the Midwest than that of F. M. Wilson (pp. 141-145), who is one of Iowa's foremost experts on swine diseases. Battling for years in a congested swine population where everything in swine breeding varies from excellent to bad, Wilson was able to tell the world just what to do when enteritis shows its ugly teeth in the hog lot. When the disease takes the form of blacktongue or beriberi, it is time to look into the ration, and where and when sanitation is poor, as is the case on many farms, the treatment must be a bird of another feather. Just do what the physician would do for cholera or typhoid fever: Pure water supply, proper disposal of excrement, emollient nourishment and, when appropriate, specific biological preventives.

APPLICATIONS

The success of the membership drive now being conducted by the central office with the coöperation of the resident secretaries is evidenced by the 72 applications listed below—an all-time record for the month of March.

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The amount that should accompany an application filed this month is $9.16. This includes membership fee and dues to January 1, 1941, including subscription to the JOURNAL.

CLINICAL DATA

Primary Mesothelioblastoma of the Epicardium of a Dog

By WAYNE H. RISER, D.V.M.
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TUMOROUS growths in which the cells are mesothelial in nature are reported only rarely in veterinary literature. Even in the human subject they are seldom encountered. Authors seldom devote over a few paragraphs to them in standard texts on tumors. This type of tumor in man was described histologically in 1870 by Wagner[1] under the name "tuberkelaehnliche lymphadenome." He believed the condition to be a pseudotuberculosis infection. Later, Schultz,[2] examining Wagner's case, described it as endothelial carcinoma. Since then, several human cases have been reported, often as dissertations, under a variety of names.

There is no general agreement, among authorities, as to the histogenesis and classification of this type of tumor. The majority of those who record cases seem anxious to prove that the condition is really one of primary carcinoma of the lung or bronchus which has spread to some of the adjoining tissue. In those cases where no primary tumor can be found in the lung, some investigators assume that the growth is small and has been overlooked. Opinion has been divided as to whether they fall in the group of carcinoma or sarcoma, or should be placed in a separate group of endothelioma. Ewing[3] urges that we consider the behavior of the neoplastic cells rather than the embryological derivation. It is not the purpose of this paper to enter into the controversy but, rather, to record the writer's findings in this case with the hope that it may help to establish a better understanding of the condition.

Jeannette D. Throckmorton, M.D., of the Iowa State medical library, was kind enough to review both veterinary and medical literature for reports of tumors that were mesothelial in character. She found reports of tumors of this kind to be very rare in both veterinary and medical literature. Feldman[4] reported a mesothelioblastoma of the pleura in a horse. In the same report he called attention to the fact that Kingsley found a similar type of tumor in the lung of a dog.

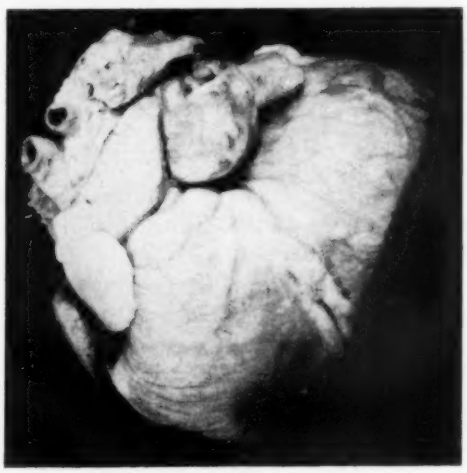


Fig. 1. Tumor in region of coronary groove on left side of heart.

Feldman,[5] in his text of a later date, reported the finding mesotheliomas once in the horse and twice in the cow. He recorded seven references of like tumors having been reported between 1902 and 1931.

REPORT OF A CASE

A male Boston Terrier, aged 7 years, was brought to the hospital suffering with a jaundice condition associated with severe

vomiting and extreme dehydration. When the case was presented, the owner reported that the dog had been in apparently excellent health until three days previously. The animal died the following day as a result of pulmonary hemorrhage.

The writer will not describe the lesions causing the illness and death of the animal because it is felt they were in no way related to the tumor being reported in this paper.

A routine autopsy was performed after death. A pedunculated grayish-yellow tumor measuring 2 by 5 cm. (0.8 by 2 in.) was found on the left side of the heart. The tumor was smooth and rounded, with an occasional yellowish spot, which gave it a fat-like appearance. It was rather firm and was strongly attached to the epicardium in the region of the coronary groove anterior to the auricle (fig. 1).

The other organs were carefully examined for any tumorous growths. They failed, however, to reveal any abnormal nodular units of tissue. It was impossible to make a satisfactory examination of the lungs because of the diffuse hemorrhage in the lung tissue.

The heart with the tumor intact was fixed in 10 per cent formalin. A small piece of the tumor was embedded in paraffin, sectioned and stained with hematoxylin and eosin.

Microscopic Examination.—The tumor contained much fibrous tissue stroma. This connective tissue was rather loosely arranged and held the tumor cells in clusters by a fine, lacy network of fibrils. The morphology of the cells and the structure of the tumor were variable. In some areas the cells took a definite alveolar arrangement while in other areas there were rows of tumor cells interspersed by banks of connective tissue.

The cells were round, oval, spindle and elongated and were markedly large. A combination of the four shapes of cells was seen throughout the tumor. The round and spindle cells predominated and gave evidence of a rapidly proliferating tumor. An occasional giant multinucleated form occurred. The cytoplasm, which was fairly granular, was abundant, with nuclei which were large and hyperchromatic. An occasional nucleolus was present. The cytoplasm stained rather uniformly and had an eosinophilic tint.

Mitotic figures were numerous. Du Bray and Rosson[6] are of the opinion that metastases are uncommon in this type of tumor. Invasion occurs by direct extension through the lymphatics and implantation metastases are found frequently on the surface of the organs lying in the cavity in which the primary growth is found.

Diagnosis.—The cells were somewhat epithelial-like and might have been mistaken for a secondary carcinoma. The writer, however, does not feel justified in calling the tumor a carcinoma, because the surface cells and not the subserous cells gave rise to the tumor. The tumor was located on the serosa and the character of the arrangement of the cells and stroma should suggest a strong possibility of a mesothelioma. The writer prefers to call the tumor a primary mesothelioblastoma. This decision was made from the description and diagnosis given in the writings of Birnbaum,[7] Du Bray and Rosson,[6] Feldman,[5] Heise and Trudeau,[8] Geschichter,[9] Shelburne,[10] Vitkus[11] and Zeckwer.[12]

DISCUSSION

This case was of particular interest because a review of the veterinary literature revealed no reports of mesothelioma of the heart or pericardium. The medical literature revealed 60 primary tumors of the heart and only six were recorded to be mesotheliomas; it did not, however, reveal a single report of a neoplasm on the epicardial surface of the heart. The writer believes from these findings that this neoplasm is one of the rarest of tumor formations. Many are of the opinion that the only injurious effect upon the body is mechanical in nature.

The immature cell forms of the tumor found in the section leads the writer to believe that the tumor was growing rapidly, and if the dog had not died from the other causes, he would have experienced

much distress and eventually died as the result of this tumor.

References

[1]Wagner, E.: Das tuberkelahnliche lymphadenom. Arch. f. Heilk., xi (1870), p. 497.
[2]Schultz, R.: Das endothelcarcinom. Arch. f. Heilk., xvii (1876), p. 1.
[3]Ewing, J.: Neoplastic Diseases (W. B. Saunders Co., Philadelphia, 1928), 3rd ed., p. 811.
[4]Feldman, W. H.: Mesotelioblastoma of the pleura. Jour. A.V.M.A., lxvi (1924-25), n. s. 19 (6), p. 754.
[5]Feldman, W. H.: Neoplasms of Domesticated Animals (W. B. Saunders Co., Philadelphia, 1932), p. 195.
[6]DuBray, E. S., and Roston, F. B.: Mesothelioma of pleura. Arch. Int. Med., xxvi (1920), 6, p. 716.
[7]Birnbaum, G. L.: Primary mesothelioma of the pleura. Bul. of Sea View Hosp., iv (1939), 4, p. 462.
[8]Heise, F. H., and Trudeau, F. B.: Primary pleural mesothelioma. Amer. Rev. Tuber., xvi (1927), 1, p. 92.
[9]Geschichter, C. F.: Mesothelial tumors. Amer. Jour. Cancer, xxvi (1936), 2, p. 378.
[10]Shelburne, S. A.: Primary tumors of the heart. Ann. Int. Med., ix (1935), 3, p. 349.
[11]Vitkus, J. T.: Pleural mesothelioma. Ohio State Med. Jour., xxxiii (1937), 9, p. 1007.
[12]Zeckwer, I. T.: Mesothelioma of the pleura. Arch. Int. Med., xxxiv (1924), 2, p. 191.

The Suprarenal Cortex

The cortex of the suprarenal glands (capsules) is indispensable to life. Complete extirpation of these glands leads to apathy, myasthenia, subnormal temperature and death in several days, provided none of the cortex remains to hypertrophy and act as an accessory gland. Extirpation of the cortex is always fatal. Its effects are multiple and interesting. Extreme muscular weakness is characteristic. It is one of the cardinal signs of Addison's disease. The decapsulated subject is immediately fatigued and quickly loses muscular glycogen. Resistance to disease and chemical intoxicants is diminished. Exposed to contagions, experimental subjects deprived of the cortex succumb rapidly in the presence of controls which survive. The effect on caloric metabolism is profound. Exposed to cold, the temperature and the arterial tension drop rapidly. Variations in the composition of the blood and urine ensue. The excretion of potassium, sodium chloride and the volume of blood are decreased. Blood acidosis through depredation of bases develops along with a certain degree of hypoglycemia and hyperthiemia associated with hypoglutathionemia.

In 1927, two American biologists succeeded in preparing an extract of the cortex which prevented death in decapsulated animals and, more recently, better extracts which give brilliant results in the treatment of Addison's disease have been available. The subject is one to bear in mind in the practice of veterinary medicine. What seems complex and not understandable today may become clear tomorrow. The fact that grave infections and intoxications make deep impressions upon the cortex of the adrenals and that the phenomena following decapsulation correspond roughly to the symptoms of many diseases may lead to the recognition of definite syndromes which will suggest the resort to corticosterone therapy. The cortex contains important quantities of vitamin C, a factor that still remains inexplicable in animal medicine. Added to the extract of the cortex, ascorbic acid prolongs the life of laboratory animals 20 to 50 hours following a fatal dose of diphtheria toxin. However, not a single instance of spontaneous suprarenal-cortex trouble has been demonstrated with certainty in animals.

Reference

Pierre: Endocrinologie surrenale. Rev. de Méd. Vét., xci (May 1939), pp. 241-271.

Heartworm Transmission

Dogs in the vicinity of Boston, Mass., harbor *Dirofilaria immitis*, as shown by four of 32 dogs in one kennel. The infected dogs included two Cocker Spaniels, one Fox Terrier and one Russian Wolf Hound, all native to the vicinity.

Transmission of the parasite was shown by six local species of mosquitoes. No evidence was found of transmission by fleas. Stable flies ingest larvae but it is problematical whether they transmit the parasite. (*J. H. Phillips. Studies on the Transmission of Dirofilaria Immitis in Massachusetts. American Journal of Hygiene, section D, xxix, 1939, pp. 121-129.*)

Meat is tenderized 19 times quicker than by hanging by the use of warmth and ultraviolet rays.

The Use of a Colloidal Suspension of Kaolin in Alumina Gel in the Treatment of Intestinal Inflammation in Dogs

By J. D. MANGES, D.V.M.
Tacoma, Wash.

KAOLIN has long been used in the treatment of dysentery and other conditions for its effect in adsorbing bacteria and irritant substances.[1, 2, 3, 4] Adsorption is an important therapeutic principle and may be defined as the mechanism by which substances are attached to the surface of the adsorbing material. Obviously, the greater the surface exposed, the greater will be the adsorption. For this reason, the dispersion of kaolin in aluminum hydroxide gel, which is a related mineral substance, greatly enhances the adsorptive effect.

In the series of 26 cases summarized in this report the principle of adsorption has been utilized in treating intestinal inflammation in dogs by the use of a colloidal suspension of kaolin in alumina gel. In using the material, seven parts of the colloidal suspension of kaolin was mixed with one part of mineral oil. This procedure avoided the constipation which sometimes occurs when the mineral oil is omitted.

In conjunction with treatment by the colloidal suspension of kaolin in alumina gel, appropriate cases were treated with other medication, such as normal saline, dextrose, stimulants, laxatives, cathartics and high and low enemas, as well as various blood antiseptics to treat the toxemias and septicemias. In vomiting dogs, the colloidal kaolin suspension was given every half hour in doses of 30 Gm. (1 oz.) until the vomiting ceased. After vomiting ceased, or if vomiting was not present, the dose was 30 Gm. every hour for dogs up to 11 kg. (25 lbs.) and 30 to 90 Gm. per hour for larger dogs.

In cases of enteritis, enemas consisting of the colloidal suspension of kaolin mixed with an equal volume of mineral oil were beneficial. These enemas were given two or three times daily, and not more than 60 cc. (2 oz.) was injected at one time. These enemas must be injected slowly at body temperature to obtain maximum retention.

Case summary.

DIAGNOSIS	NO. OF CASES	SYMPTOMS	OUTCOME
Gastroenteritis	8	Vomiting and diarrhea, blood in vomitus and stools	All recovered
Poisoning—food or undetermined	6	Vomiting, no appetite, thirst, abdominal pain, blood in bowels	5 recovered, 1 died (moribund when first seen)
Gastritis	3	Vomiting, dehydrated, no appetite	All recovered
Bloody bowels	3	Blood in stools and passing from bowels, no appetite	2 recovered, 1 died (moribund when seen, had been bleeding severely)
Bowel infection	2	Vomiting, blood passing from bowels, no appetite	All recovered
Infectious enteritis	2	Watery stools containing blood, no appetite	All recovered
Hemorrhagic enteritis	1	No appetite, complete cachexia	Euthanasia
Irritated rectum	1	Blood from rectum	Recovered

It also was found desirable to wash the animals through with the same solution used for the enema in all cases where it was not expedient to wait until the kaolin suspension given by the mouth had had time to pass through.

The colloidal suspension of kaolin in alumina gel when administered by the mouth can usually be found in the feces in twelve to 18 hours and, in a few cases, in as short a time as eight hours. In cases which were autopsied the examination revealed a fair amount of the colloidal suspension distributed evenly through the bowel as far as the rectum.

The cases which are summarized cover, with one or two exceptions, only those animals which were hospitalized and treated by the writer. From my observations on these cases, I believe that the colloidal suspension of kaolin in alumina gel is indicated in all cases of inflammation of the bowel, regardless of cause, and when used in conjunction with the substances described above, and with proper attention to the removal of the cause, it gives highly satisfactory results.

References

[1]Braafladt, L. H.: The effect of kaolin on the intestinal flora in normal and pathologic conditions. Jour. Inf. Dis., xxxiii (November 1923), p. 434.

[2]Swalm, W. A.: Aluminum silicate (kaolin) with aluminum hydroxide in certain intestinal disorders. Med. Rec., cxl (July 4, 1934), p. 26; and cxl (July 18, 1934), p. 68.

[3]Fradkin, W. Z.: The control of rectal bleeding in the convalescent ulcerative colitis patient. Jour. Lab. & Clin. Med., xxii (June 1937), p. 896.

[4]Eyerly, J. B., and Breuhaus, H. C.: Further studies in the treatment of ulcerative colitis with aluminum hydroxide and kaolin. Illinois Med. Jour., lxxiv (December 1938), p. 534.

Local Use of Vitamin A

Cod liver oil, owing to its high vitamin content (A and D) is said to be utilitarian in the treatment of open traumatic injury. It is one of the ingredients of numerous "healing ointments" used in veterinary medicine on the grounds that it steps up the proliferation of formative tissue and subsequent cicatrization. This is a property manifestly difficult to prove experimentally. Unchecked clinical observations (upon which the use of the majority of drugs is founded) are invoked in defense of its medicinal value.

It is reported* that vitamin A is an ideal local application in traumatic injury and trophic lesions of the cornea. Applied to the lesioned cornea, vitamin A promotes epithelization and relieves pain. An oil containing 1,000 I.U. per cubic centimeter is recommended for the purpose. For palpebral wounds where suturing is not necessary, a petrolatum ointment containing 500 I.U. per gram is employed.

For grave conjunctival infections, sterile milk subcutaneously, sulfanilamide orally, and a vitamin A preparation of the proper strength are excellent therapeutics. The report (*loc. cit.*) goes on to say that oleaginous suspensions of vitamin A are absolutely indicated in ocular injuries of industry and war, torpid ulcers, recurrent erosions, bulbous, neuropathic and filamentous keratitis, corneal degeneration of obscure origin, *et al.* In short, this type of local treatment is held out as a veritable panacea for superficial eye injury.

Infections No Longer Strictly Regional

The pathways of travel for man and material across the boundaries of the temperate zones impose new problems on the science of medicine. Serene contentment about infections in far-away places is no longer safe. A pathogen may be quite as comfortable in the warm body of an animal in North Dakota as in Brazil and, if means of mutation exist, it can be quite ubiquitous. On that account alone, those engaged in controlling diseases of man, animals and plants must be far-seeing and watchful. In fact, diseases we do not recognize in the patients which file before our eyes may be more numerous than we suspect.

Loeffler and Schutz discovered the bacillus of glanders. They published a preliminary report on their discovery in 1882 and an elaborate monograph on the subject in 1886.

*Abstract from Archives of Ophthalmology, xxii, November 1939, p. 727, in The Journal of the American Medical Association, cxii, December 30, 1939, p. 2452.

Aural Hygiene

By T. B. CROWE, M.D.C.

Chicago, Ill.

THE DEVELOPMENT of small animal practice has focused attention to a body orifice that has received but scant attention in animal pathology, namely, the external acoustic meatus (= meatus acusticus externus), commonly called the external auditory canal, the path of sound to the ear drum (= membrana tympani).

The canal is composed of two parts: Cartilaginous and osseous. The former is made up by folding of the conchal and annular cartilages of the auris externa and completed medially to the drum by the external acoustic process of the petrous temporal bone. The lining is skin extended from that of the conchal cartilage (= auris externus). The cartilaginous or lateral portion contains numerous fine hairs and a large number of wax glands (= glandulae ceruminosea). The osseous or medial portion contains but few (if any) hairs and is but sparsely studded with glands.

Anatomically, therefore, the structure of which we speak is composed of cartilage, bone, skin, hairs, and wax-secreting glands, arranged along the wall of a canal interrupted by obstructing anfractuosity designed to block admission of particulate material to the sound-receiving mechanism within and, thus, to safeguard the sense of hearing against the many exterior vicissitudes to which animals are constantly exposed. In this respect the meatus acusticus externus of dogs and other mammals is truly "fearfully and wonderfully made." The wonder is that this apparently wide-open channel is not continuously choked with foreign matter and the issues of reactions to them. The main deficiency of this remarkable canal is the lack of sufficient protection at the depths where wax glands are sparse, and material surmounting the cartilaginous portion can collect and cause the troubles we are inclined to classify a specific disease-canker.

Since canker is not caused by a specific organism, external otitis is the better name. This common affliction of dogs answers precisely to the description of a continuous local irritation brought about by the nesting of irritant material (wax, dirt, bacteria, exudates, acarian mites). The troublesome lesions which complicate matters are but the expected reaction of such a structure to these foreign inhabitants.

Where these facts are made the basis of treatment, ear canker is more easily conquered. On the other hand, where harsh irrigations and swabbings with irritant drugs are employed, many patients pass from one doctor to another in the hope of finding the cure. The medial portion of the canal is an exceedingly delicate locale. Its membranous structure is more sensitive to microbicides than the bacteria one tries in vain to destroy. The result is more exudate, more detritus, more unnatural granulation and more chronicity than if the trouble had been either left alone or handled more gently.

But, what seems quite as important in the care of dogs is more and better attention to this canal, practiced along with the frequent bathing and extensive grooming, and habitual tartar scraping to which fine dogs are subjected. To pass over this filthy canal, often diseased, in executing the customary hygienic measures required to keep dogs in the bloom of good health, is a neglect of considerable importance. No dog is "well groomed" whose external auditory canal is not clean and not functioning in a normal fashion. The dehydrating glycerol, auralgan, is useful for that purpose, since it quickly captures the harmful fluids of the canal without interference with the protective secretions. Thus, in addition to the use of this popular "ear remedy" for external otitis in human and veterinary medicine, its prophylactic properties should not be overlooked in the general care of a dog or cat.

Anthrax in Minks*

By GEO. W. STILES and C. L. DAVIS, D.V.M.
Denver, Colo.

THE RECOGNITION of anthrax in domestic animals is a common experience in veterinary practice; however, its appearance in wild or domesticated fur-bearing animals is seldom observed. The earliest available reports (1931) of anthrax in minks came from western Germany, where Sprehn and Albrecht[1] reported outbreaks of anthrax on mink farms. On the first ranch eight of twelve minks died, and on the second ranch 18 of 25 died. From the third lot one carcass only was received, without history. The mink losses on the first ranch occurred two or three days following the feeding of anthrax-infected beef.

Meyer[2] cited the loss of 15 minks, two males and thirteen females, from anthrax on two farms having a total of 30 head. These animals were fed meat from an emergency-slaughtered cow, the flesh of which was proved by bacteriological examination to contain anthrax bacilli. Grini[3] diagnosed anthrax in four minks fed meat from a cow supposed to have died naturally. In his thesis on the pathology of mink disease, Schloesser[4] cited a case of anthrax in silver foxes and stated that this disease has been known in the wild red foxes of Europe for a long time.

Anthrax in minks has been reported recently in the United States. Pinkerton,[5] in St. Louis, diagnosed this malady in minks from two ranches. Both outbreaks were ascribed to the eating of infected horse meat. In Oregon, Howarth and Seghetti[6] found anthrax in four dead minks from an area supposedly free from this organism. The exact source of the infection was undetermined.

Greener[7] cited the records of the chief veterinary officer of the ministry of agriculture of Great Britain, which show that 16 minks and one ferret died of anthrax during January 1936 on a farm in Kent. Seven minks died from this malady during July 1937 in East Sussex. Two raccoons and two badgers died from anthrax in Kent. This investigator diagnosed six outbreaks of anthrax in minks during 1938 with a mortality of about 25 per cent among 1,290 animals. Greener also reported the death from anthrax of two silver foxes. Infected meat was ascribed as the source of infection in a number of the cases.

AN OUTBREAK IN THE DENVER AREA

An outbreak of anthrax occurred in the vicinity of Denver, Colo., during September 1939. A sudden onset of illness developed in three different mink ranches at the same time. All three ranchers obtained their meat supply from the same source. On the premises of the first ranch eleven minks out of 500 died in five days. On the second ranch, three died out of 86, and on the third ranch two died out of 36—a total death loss of 16 from the three ranches, representing 622 minks. Then of the dead minks were submitted to the branch pathological laboratory of the U. S. bureau of animal industry for diagnosis.

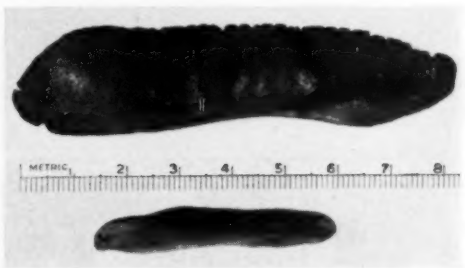


Fig. 1. Mink spleens. Above, one from a case of anthrax.
Below, a normal spleen. The animals from which these were obtained were both 4 months of age.

LABORATORY FINDINGS

The first three minks brought for examination had been pelted. The carcasses ap-

*From the Denver branch pathological laboratory, Bureau of Animal Industry, U. S. Department of Agriculture.

peared well nourished, with no evidence of external injury. A few drops of blood were observed issuing from the nose of one animal. A male mink showed blood-stained urine dribbling from the urethra.

Each of the ten minks autopsied showed only slight pathological differences. All had engorged blood vessels, filled with dark, semicoagulated blood. On gross inspection the hearts appeared free of congestion.

Fig. 2. Stained smear from the spleen of an infected mink. Note the square-end rods.

The spleens were enlarged, darkened, pulpy, and easily torn (fig. 1). Some animals showed a moderate gastroenteritis and two cases revealed distinctly bloody urine.

Our suspicions as to either anthrax or paratyphoid infection were aroused immediately upon observing the character of the spleens. Microscopic examination of smears from spleen tissue revealed typical anthrax bacilli (fig. 2). Cultures were prepared from the viscera, and pure strains of anthrax organisms were obtained. The diagnosis of anthrax was confirmed by laboratory-animal inoculations.

Dietary of Minks on Infected Premises

The food used for the minks, prior to the appearance of anthrax on the three ranches involved, consisted of horse meat, cold storage jack rabbits, tripe, lungs, a little green bone, and cereal. The percentages of each ingredient varied slightly from day to day. About 375 kg. (825 lbs.) of meat products were prepared weekly for mink feed. Foxes also ate of the same general supply without ill effect.

During the probable incubation period and before the occurrence of the anthrax outbreak, two horses and two cows had been slaughtered and their meat added to that held in the cooler. Bacteriological examination, including guinea pig inoculation, of the salted hide from one of the horses which was slaughtered, because of illness, gave negative evidence for anthrax. What bactericidal effect the salt may have exercised during the ten-day pickling process remains undetermined.

One of the cows killed had a "prolapsed uterus." The history of the other cow and horse was not obtained. Composite samples of blood from the slaughtered cows and horses which were fed during this period gave negative results for anthrax through laboratory-animal inoculation. Various organisms were recovered from this material. Even though anthrax bacilli may have been present some days previously, the overgrowth of saprophytic types prohibited their being identified.

Inspection of Premises

During the occurrence of anthrax on these mink ranches, large numbers of flies were seen. When anthrax was proved to be the cause of death in the minks, a campaign of spraying and trapping was started at once. The daily disposal of droppings and other sanitary measures also were inaugurated. Dead flies and specimens of feces collected from pens where minks died failed to yield anthrax bacilli from laboratory examination and guinea pig inoculation.

Discussion

The appearance of anthrax in three mink ranches in a community where this disease was unknown in domestic animals caused much discussion as to its probable origin. The jack rabbits came from various localities, including areas where anthrax is known to be epizoötic. Also, the horse meat and beef came from animals slaughtered

in different places, some even from outside Colorado.

Feeding meat from questionable sources to fur-bearing animals is a dangerous practice. Every safeguard should be taken to insure a wholesome dietary for minks and foxes by slaughtering only healthy food-producing animals under strict sanitary conditions.

Since only 16 minks died from a total of 622 head, it would appear that a small quantity of food was infected with anthrax organisms in sufficient numbers to cause death.

SUMMARY

An outbreak of anthrax in minks occurred during September 1939 in the region of Denver, Colo., which resulted in the death of 16 animals in three ranches representing 622 head.

The diagnosis of anthrax in these minks was definitely established by laboratory examinations, including guinea pig inoculations.

The definite source of the infection was not determined, although some of the meat fed the minks was suspected as the probable cause. The meat used came from jack rabbits, horses and cows. Some of the horses and cows were known to be sick at the time of slaughter, yet their meat was fed to valuable fur-bearing animals.

The practice of feeding foxes and minks the flesh from animals known to be sick at the time of slaughter is a dangerous procedure. Inspection by competent veterinarians should be provided for all animals before and after slaughter, and the meat should be preserved by adequate refrigeration.

References

[1]Sprehn, C., and Albrecht, O.: Kurz mitteilung über einige sektionsergebnisse aus der pelztierpraxis. Deut. Tierärztl. Wochnschr., xxxix (Aug. 8, 1931), 32, pp. 499-502.

[2]Meyer, O.: Milzbrandseuche in einer peltzierfarm. Berl. Tierärztl. Wochnschr., xlvii (Oct. 16, 1931), 42, p. 686.

[3]Grini, O.: Die krankheiten des mink. Berechtigte übertragung aus dem norwegischen von tierärzt Eugen Bass. Pelztierzucht, ix (Jan. 3, Feb. 1, March 1, 1933), 1, pp. 1-5; 2, pp. 23-38; 3, pp. 39-42.

[4]Schloesser, M.: Pathologie du vison. Thèse, Alfort (1935).

[5]Pinkerton, H.: An outbreak of anthrax infection in minks with infection of a ranch owner. Jour. Amer. Med. Assn., cxii (March 1939), 12, pp. 1148-1149.

[6]Howarth, C. R., and Seghetti, L.: Anthrax in farm-raised mink in Oregon. Jour. A.V.M.A., xciv (1939), n. s. 47 (4), pp. 433-434.

[7]Greener, A. W.: Anthrax in mink (*Mustela vison*). Jour. Hyg., xxxix (March 1939), 2, pp. 149-153.

Sarcosporidiosis of the Tongue and Lip in a Horse

THE SUBJECT was an aged gelding with an enlarged tongue and lip of four-month duration. The portion of the tongue anterior to the phrenum was greatly enlarged (see photograph) and, on palpation, firm and inelastic. The animal was emaciated but was able to partake of food and drink sufficient to sustain life.

The affected portion of the tongue was amputated and a laboratory examination revealed numerous sarcosporidia.

Tongue of the affected horse.

Six months following the operation, the owner reported that the horse had gained 114 kg. (250 lbs.) but that he was still not in his regular flesh. The swelling had receded somewhat but was still very noticeable. The animal had worked for about ten hours at different times in a single cultivator, but showed no spirit. "I do not think it pays to keep him," the owner stated, "and, although I still have him on pasture, will dispose of him shortly."—*John D. Beck, V.M.D., School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa.*

Medical corps units of mobile armies are now equipped with x-ray apparatus for use on the battlefields.

Studies on Thysanosoma Actinioides*

By F. X. GASSNER, D.V.M., and FRANK THORP, JR., D.V.M., M.Sc., Ph.D.
Fort Collins, Colo.

ALTHOUGH the existence of the parasite, *Thysanosoma actinioides,* has been known for over a century, little information is present in the literature regarding its life cycle. Important literature dealing with this tapeworm is reviewed in the publications of Stiles and Hassall,[1] Curtice[2] and Hall.[3, 4] The cestode is described in the well-known textbooks of parasitology.

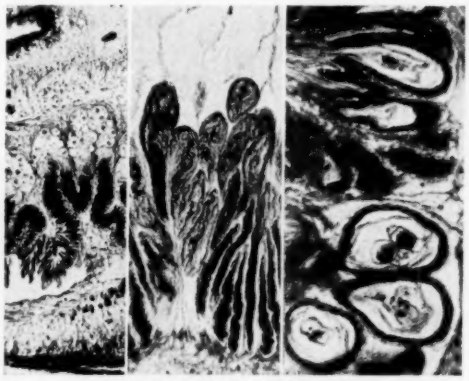


Fig. 1. (left). Development of ova in uterine tube. Fig. 2 (center). Parauterine pouch. Fig. 3 (right). Embryonating ova in parauterine pouches.

Although the parasite is only slightly, if at all, pathogenic for feedlot lambs, it is of importance in that livers from such animals infested with this worm are condemned.

During the fall of 1938, three worm specimens were sent to the laboratory for identification. During the examination of one of these specimens, which was identified as a segment of *T. actinioides,* six encapsulated oval bodies were observed under the same cover slip outside of the proglottid. Several of these oval bodies or embryonated egg cases were subjected to micro-dissection. The ova could be liberated readily. The terminal segments of more than 100 worms from the stock of preserved specimens were systematically examined for embryonated egg cases, but none was found. From the terminal segments of a few of these worms, single ova were obtained. These structures proved to be morphologically identical with those observed by Newsom[5] and which have been photographed by Newsom and included in a bulletin by Newsom and Thorp.[6] Since these embryonated egg cases were not observed in the segments, it was impossible at that time to prove their direct relationship to this cestode.

In examinations of lamb feces for parasite ova, only one embryonated egg case was found during 1939. In the small intestine of the lamb whose feces showed the egg case, fringed tapeworms were present at autopsy. In the contents of the large intestines, a few detached segments were found which contained embryonated egg cases. From then on, when this cestode was encountered in the small intestines of lambs, the contents of both intestines were separated and repeatedly washed and decanted. A few segments of the fringed tapeworm were recovered by this procedure from either the small or the large intestine, or from both. These segments contained the embryonated egg cases. The cases were not found in the terminal segments of entire worms taken from the small intestines. However, a critical examination of the uterine tract of the terminal segments of some worms revealed ova in various stages of development, but no embryonated egg cases. This procedure has been repeated 20 times, and 55 segments have been recovered.

The ova are presumably fertilized within the convoluted uterine tube (fig. 1). At or after cleavage, the developing embryos pass into the parauterine pouches, four to six of which are connected by a slender neck with the anterior curvature of the uterine convolutions (figs. 2 and 3). Upon maturation the parauterine pouches con-

*From the Colorado Experiment Station.

tain seven to twelve embryonating ova. The slender necks of some of these pouches are severed at the junction with the uterus proper and, probably as a result of muscular contraction and/or increased volume, concentrate in the anterior portion of the segment, which seems to be the point of least resistance. It has been repeatedly observed that the embryonating ova are not discharged from the segment singly but remain in these pouches which finally constitute the embryonated egg cases.

The segments attain various shapes—square, cylindrical or semilunar—depending upon the degree of gravidity, and are not ordinarily detached, unless fully gravid. The fully gravid ones become opaque as a result of the concentrating of the embryonated egg cases. When a gravid segment is placed upon a slide, covered gently with a cover slip, and then examined with the binocular microscope, the embryonated egg cases may be observed in and above the reproductive system. If gentle pressure is applied with a teasing needle over the segment, these egg capsules are observed to leave an aperture located in the anterior median portion. The egg cases apparently are not discharged through either of the lateral genital pores, as has been presumed. They emerge from this aperture singly and by their long axis.

The egg cases containing the ova are encapsulated forms with one end rounded, the other end terminating in a frayed, tail-like structure (neck of parauterine pouch). The capsule is composed of fibrous connective tissue. The outside measurements of ten egg cases were 145.8 to 267.7 μ by 110.5 to 123.2 μ. The cavity containing the ova measured 75.5 to 96.2 μ by 50.8 to 60.6 μ.

Structurally the embryonated ova show the embryo within a single shell which measures 11.55 μ and is remarkably uniform in size. The hexacanth is distinctly visible and the hooklets are, on the average, 3.85 μ in length. Surrounding the embryo proper is a colorless, corona-like structure which is of nonrefractile, transparent material. This extra embryonic covering is very resistant to pressure and shows only a faint affinity for basic stain.

Further research is being carried on at the present time.

References

[1]Stiles, C. W., and Hassal, A.: A revision of the adult cestodes of cattle, sheep and allied animals. U. S. Dept. Agri., Bur. Ani. Ind. Bul. 4 (1893).

[2]Curtice, C.: The tapeworm disease of sheep of the western plains. Ann. Rpt., Bur. Ani. Ind., U. S. Dept. Agri. (1889), pp. 167-186.

[3]Hall, M. C.: Parasites and parasitic diseases of sheep. U. S. Dept. Agri. Farmers Bul. 1330 (1931, 1932).

[4]Hall, M. C.: The discharge of eggs from *T. actinioides*. Proc. Helminth. Soc. Washington, i (1934), 1, pp. 6-7.

[5]Newsom, I. E.: Personal communication (1939).

[6]Newsom, I. E., and Thorp, Jr., F.: Lamb diseases in Colorado feedlots. Colo. Exp. Sta. Bul. 448 (1938).

Staphylococcic Infections

OBSERVATIONS, clinical and experimental, on the medicinal value of sulfanilamide and its derivatives in streptomycoses are numerous and precise. Yet, the same has not been claimed in animal medicine for these remarkable drugs in the treatment of staphylococcic infections. The teaching which would tend to restrict sulfamidotherapy to the streptococcic septicemias does not, however, seem justifiable. Personal observations in hospitals of the Chicago district and a glance through the literature of the past two years seem to indicate that certain staphylococcic septicemias do yield to the action of sulfanilamide and prontosil quite as promptly as do streptococcic types.

By having selected only cases of proved staphylococcic origin which, without question of doubt, were cured because of the treatment, there has been found sufficient reason for recommending sulfanilamide or prontosil for that class of infections in animals, and with quite as much audacity as for the various streptomycoses, provided the case is septicemic and the diagnosis correct. In general, authors emphasize the futility of sulfamidotherapy in local or dermatostaphylococcia, such as demodectic mange. The treatment is resultful only when the infection is generalized.

Thornhill, Swart and Reel[1] report two cases (in man) of staphylococcic septicemia that were cured with sulfanilamide, blood

transfusions and surgical drainage. But, inasmuch as this infection is invariably fatal, the credit goes to the drug treatment. Berthelon and Brovois,[2] who have used a sulfamide derivative in various staphylococcic infections for the past two years, always controlling the diagnosis bacteriologically, reported fine results, except in chronic cases, in mastitis in cows, bitches and rabbits. Mery[3] obtained a rapid cure of a grave case of staphylococcemia, recognized by hemoculture, in a 4-year-old dog. Henry and Guillon[4] obtained a rapid cure in a dog affected with facial anasarca—a grave staphylococcic infection associated with a high fever and subglossal adenitis.

The grave surgical infections in animals can be controlled to a remarkable extent by dosing with sulfanilamide or prontosil. Most of them are predominantly staphylococcic.

References

[1]Jour. A.M.A., cxii, Oct. 28, 1939, p. 1638.
[2]Rec. de Méd. Vét., cxv, June 1939, pp. 337-349.
[3]Les cahiers de médecine vétérinaire, April 1937, p. 121, cited by id.
[4]Bul. de l'Acad. Vét. de France, July 1938, p. 467.

Periodic Ophthalmia

To the Editor.—I shall be grateful to you for any information you can offer as to periodic ophthalmia in horses and its treatment. I realize that if the first and second attacks pass untreated, there is little that can be done. It therefore seems that the first attack must be treated vigorously in order to ward off the second and third attacks.

I have used neoarsphenamine intravenously several times with good results, but I am somewhat hesitant in resorting to it because of the swellings that sometimes follow its administration. It has occurred to me that the neoarsphenamine solution that I use is not sufficiently dilute and, thus, may be too irritating. I also have used sterile milk subcutaneously in a few cases, with comparable satisfaction. In either treatment, I always use an atropine ointment to dilate the pupil, and I keep it dilated until the eye has cleared.

I am now using an intravenous outfit in which one long tube or catheter goes through another shorter hypodermic needle. However, the long tube has two openings at the lower end which cause the solution to squirt sideways against the sides of the jugular vein, thereby injuring it. I believe that I shall trim the tube just above the two openings so that the solution will come out of the end of the tube, thus not squirting against the jugular.

I always make the injections slowly.—*F. C. M., Ohio.*

Reply.—There is no reason that arsphenamine should not be diluted to a strength two or three times weaker than the dilution usually recommended. There is no doubt whatsoever that injury to the intima is quite as often responsible for the local reactions as perivascular delivery of a part of the dose. To insure immediate mixture of the fluid with the blood, gentle and very slow delivery (as is your policy) is of paramount importance. A cannula with but an end opening is preferable.

Since much remains to be learned about the cause of periodic ophthalmia, there is no specific treatment. Arsenic for its alterative action, atropine to prevent synechia and damage to the capsule of the lens, green-colored forage for its carotene content, and sterile milk, which is reputed to affect favorably the metabolism of the cornea in horses, is the general plan of treatment at the present time.

Cat Bites

Cats harbor a saprophyte in their throats that can be isolated in pure cultures from wounds caused by their bites. The pathological manifestations, which begin to appear about two weeks after the bite, are local edema, violent local pain and a state of illness without a rise of temperature. The organism is a short, hemoglobinophilic bacillus of the Pasteurella group that is highly pathogenic for other animals and man. (*Lienze. New Points in the Pathology of Cat Bites.* [*Title translated.*] *Abst., Revue de Médecine Vétérinaire, xci, April 1939, p. 221.*)

A Case of Collyriclum Faba Infestation in a Purple Finch*

By F. R. BEAUDETTE, D.V.M.
New Brunswick, N. J.

ONE GATHERS from the literature that, in 1819, Meissner exhibited before a Swiss society certain spherical swellings taken from the skin of a European finch (*Fringilla spinus*) which were regarded as *Cysticercus cellulosa*. In 1831, however, Bremser demonstrated that the swellings contained flukes to which he gave the name *Monostomum faba*. But, in 1911, Kossack created the genus Collyriclum, and the parasite was henceforth known as *Collyriclum faba*. In that year Cole reported the occurrence of the parasite in this country. His specimens were taken from English sparrows (*Passer domesticus*) at Madison and Ripon, Wis. In 1914, Riley found the parasite on this host at Ithaca, N. Y., and in 1918, Tyzzer[1] reported it in the same host killed near Boston. Three such birds were taken in the summer of 1915, and one each in the summers of 1916 and 1917 out of several dozens killed. In 1908, Hassell found the parasite in a bluejay (*Cyanocitta cristata*) taken in Maryland. An infestation in a young robin (*Planesticus migratorius*) taken at St. Paul, Minn., in June 1919 is reported by Riley and Kernkamp.[2] Riley[3] has found infested English sparrows in various parts of Minnesota, and at Waconia, Minn., an infested bronze grackle (*Quisculus quiscula aeneus*) was found.

Ward was of the opinion that Cole's parasite differed from the European form and therefore named it *Collyriclum colei*, but in 1926, Chapin[4] examined a European form and could find no possible reason for recognizing two species.

The occurrence of this parasite in domesticated birds was reported for the first time by Riley and Kernkamp[2] in 1925. A young chicken which showed "blisters" around the vent was presented in June 1922 from a farm on Lake Minnetonka near Minneapolis. In June 1923, a turkey similarly affected was presented from a farm at Vining, Minn. A further examination of this flock showed 50 affected out of 110. All of the affected birds had had the run of the lake shore, whereas the nonaffected turkeys had been reared on higher ground. Then, a survey was made of the chicken farm found to be infected in 1922, and though it was not possible to examine young birds, several apparently fresh cysts were found on two old hens. According to Riley, an infestation in poults was reported by Marotel in 1926 as occurring at Romans in the Province of Drome, in southeastern France.

Six additional infestations in domesticated birds were reported by Riley[3] in 1931. Two of these concerned market chickens of unknown origin. Another outbreak in chickens and three in turkeys were found in a county adjacent to the one in which the infestation in turkeys was found in 1923. It is Riley's opinion that infestation occurs only in birds that have access to swampy lake shores in the early season and that English sparrows serve to disseminate the flukes. Two of the owners of infested farms actually noted infested dead sparrows on the premises. Although the life cycle of the parasite has not been worked out, Riley is of the opinion that snails and the nymphs of dragonflies, respectively, serve as intermediate hosts.

The report of McIntosh,[5] in 1935, lists two additional hosts. A crow infestation was first observed in three fledglings taken June 28, 1928, at the University of Michigan biological station, Douglas Lake, Mich., and on July 14, another infested bird was taken. From July 18-26, 1929, the infestation was found in three young and one adult. Two infested white-breasted nuthatches (*Sitta c. carolinensis*) were taken on July 2, 1928, from the north shore of Douglas Lake. Infestations in crows, the

*Journal series paper of the New Jersey Agricultural Experiment Station, department of poultry husbandry.

bluejay and the red-winged blackbird have also been found by Riley.[6]

CASE REPORT

A live female purple finch was sent to us from Demarest in Bergen county, N. J., by B. S. Bowdish, secretary of the New Jersey Audubon Society, but arrived dead on May 2, 1939. The letter of transmittal contained no history. The bird bore band (139) 65099. Just below the vent were two hard, cyst-like structures, each showing a faintly visible pore. The cysts measured about 6 mm. in diameter. As the cysts were suspected of containing flukes, great care was used in cutting through the tough wall. From each cyst two flukes were removed and tentatively identified as *Collyriclum faba*. We are indebted to P. D. Harwood of the zoölogical division of the federal bureau of animal industry for the positive diagnosis of the flukes as *Collyriclum faba*. The material has been deposited in the U. S. National Museum helminth collection and assigned Catalogue 40619. A blood smear of the finch also revealed a heavy infestation of microfilariae.

References

[1]Tyzzer, E. E.: A monostome of the genus Collyriclum occurring on the European sparrow, with observations on the development of the ovum. Jour. Med. Res., xxxviii (May 1917), n. s. 33 (2), pp. 267-292.

[2]Riley, W. A., and Kernkamp, H. C. H.: Flukes of the genus Collyriclum as parasites of turkeys and chickens. Jour. A.V.M.A., lxiv (1924), n. s. 17 (5), pp. 591-599.

[3]Riley, W. A.: *Collyriclum faba* as a parasite of poultry. Poultry Sci., x (May 1, 1931), pp. 204-207.

[4]Chapin, E. A.: Jour. Parasitol., xiii (September 1926), p. 90.

[5]McIntosh, A.: New host records of parasites. Proc. Helminth. Soc. of Washington, ii (July 1935), p. 80.

[6]Riley, W. A.: Personal communication.

Avitaminoses are not necessarily due to insufficient intake of the vitamin concerned. Depressed physiological processes and pathological conditions may cause non-utilization or destruction of stored vitamins. On this account, the use of vitamins in the treatment of disease is a study of capital importance. Any tissue or organ of the body may be stricken by a vitamin deficiency.

Sodium Hyposulfite (= Sodium Thiosulfate)

Sodium hyposulfite (= sodium thiosulfate) has been employed for a long time in the treatment of septic conditions, particularly mastitis and gastrointestinal toxicoses. Except that it was said to liberate sulfurous acid or sulfur oxide, no explanation was given for its use. With the coming of more active intestinal antiseptics it was no longer commonly used. The discovery of its antidotal power in hydrocyanic poisoning, however, again brought this common sodium salt into prominence in veterinary medicine. For that purpose, doses of 24 to 40 cc. (6 to 10 dr.) of a 30 per cent solution are employed intravenously. This dosage is used in combination with sodium nitrite and methylene blue, and for cattle this may be supplemented with a 120-cc. (4-oz.) dose given by the mouth.

While various plants are cyanogenetic, wilted sorghums used to supplement pastures are among the common causes of the intoxication, though cattle overfed with other forms of roughage develop toxicoses akin to hydrocyanic poisoning. In certain parts of Ontario where rape is grown for fall pasturing, cattle develop a digestive disturbance that is similar to the trouble arising from overfeeding with green corn. With the rumen full of fermenting rape, the animal becomes depressed, grunts with each respiration and becomes semicomatose, the victim of a toxic principle.

The author cites a case caused by eating green corn and frozen and wilted turnips wherein intravenous doses of 500 cc. (1 pint) of a 30 per cent solution of this salt brought favorable responses promptly. (*R. A. McIntosh. Sodium Hyposulphite. Canadian Journal of Comparative Medicine, iii, December 1939, pp. 328-329.*)

One of the most fatal diseases of the canary (*Serinus canaria canaria*) is canary-pox, the variola of that species. It is a disease of the aviary rather than of the isolated caged bird. The mortality is 100 per cent.

SURGERY & OBSTETRICS

The Use of Silk Sutures in Veterinary Surgery

By JOHN D. GADD, V.M.D.
Towson, Md.

THE USE of buried silk sutures has been condemned so much in veterinary literature that we felt justified in investigating their use. Since we began to use silk sutures in our hospital, the end results have been appreciably better. Before the use of silk, our post-operative infection percentages were high; there were suppurations, hernias, slowly granulating wounds, long hospitalization with resultant secondary contagious infections, and other complications.

The use of silk necessitates more care from the time of preoperative preparation to the final closing of the wound. The preoperative care of the animal—that is, enemas, proper starving, fecal and urine evacuations and preanesthetic injections of atropine and morphine—is strictly supervised. We operate as a team of operator, assistant and anesthetist. Each has a definite rôle in the preoperative procedure.

The equipment, consisting of towels, drapes, masks, caps, gowns and gloves, must be in perfect shape. High-grade instruments are an essential, particularly hemostats that are not too heavy, for clamping very small vessels, and are accurate and positive in their closure. The use of intratracheal anesthesia[1] has been helpful in our procedure, since it gives a much safer anesthesia over a longer period of time. We do not "operate by the clock," and the abdomen is not closed until we are satisfied that complete hemostasis is obtained, even though more time is thus required.

Proper wound healing, that is, healing of the wound by first intention, is complicated. It depends upon the presence or absence of bacteria in the wound and the resistance of the animal. Aseptic wound healing requires, as stated above, a systematic, meticulous, preoperative, operative-room technic, careful preparation of the skin, complete hemostasis, sharp dissection, no dead spaces, no mass ligatures and, last but not least, a proper selection of suture and ligature material.

Most surgeons have been afraid to use nonabsorbable sutures and ligatures because pathologists have emphasized that foreign bodies are detrimental to tissues by virtue of possible persistent infection. This fear has been justified by the poor results which followed the incorrect use of silk.

The use of silk is not new; such suture material was in use as far back as 1870. Because it gave poor results it was discarded by many after the introduction of catgut. However, Kocher and other German surgeons continued to use it. Halsted,[2] who observed Kocher in the '80s, perceived the underlying value of the silk technic and pioneered its use at the Johns Hopkins clinic. He summarized his objections to catgut as follows:

> The relatively high cost of catgut, its bulkiness, the inconveniences attending its use and sterilization, its inadequacy, the uncertainty as to the time required for its absorption, and the reaction which it excites in a wound, induced me to discard it completely for clean wounds in the surgery both of the human subject and of animals. With the fine silk in our wounds, which for 23 years have, as a rule, been closed without drainage, suppuration almost never occurs. But catgut, even that which we have no cause to believe is not sterile, irritates the wound for some reason, perhaps because it serves as a culture medium for saprophytic organisms which are carried into it from the deep epithelium and follicles of the skin. It should be borne in

mind that during the greater part of the period of its disintegration, the catgut suture is not only not serving its purpose but is playing the rôle of necrotic tissue, of a culture medium. It is well within reason to expect that the technic may be at least so perfect when silk is employed that the wound will become infected not once in a hundred cases.

Recent clinical and experimental studies substantiate Halsted's contentions. Shambaugh and Dunphy[3] demonstrated on dogs that operative wounds repaired with silk tolerate bacterial contamination better than similar wounds repaired with catgut, that the healing of experimental suppurating wounds is not appreciably delayed by the use of the buried silk sutures, provided that a fine grade is used, that the sutures are cut close to the knot, and that continuous sutures are not employed. They also showed that experimental suppurating wounds repaired with fine silk may heal completely without removal or spontaneous discharge of the sutures. Kraissl[4] demonstrated the possibility of an allergic reaction to catgut in patients, with edema of the wound edges and disruption of abdominal wounds.

Superiority of silk over catgut is due to the following facts, according to Meleney[5]: 1) Hemostasis is better, for the silk knots do not become untied as do catgut knots. 2) The cellular fluid reaction about silk is minimal, while about catgut it is maximal; silk is almost inert in the tissues, while catgut is dead tissue which must be digested. 3) The use of silk automatically requires the surgeon to be more gentle with the tissues.

Since we are not able to immobilize dogs, with this technic of abdominal wound healing occasionally there is a serosanguineous discharge in the subcutaneous tissue. If this occurs, we nick the skin and make a small opening into the involved area. This is done alongside of the wound. Seldom do we have to open the drainage or express the discharge out again. If it is a suppurating type of discharge, the opening is made slightly larger so that the area may drain for two or three days. If the dog is sent home in 24 or 48 hours after operation, we request that it be brought in for observation every two days for several days following. In this way we are able to minimize any secondary capillary hemorrhage or possible suppuration. Some of the so-called cysts have been tapped with an 18-gauge needle, thus expressing the serosanguineous fluid. Thereafter, first-intention healing has taken place. These complications or sequels are so slight and infrequent, and so easily handled, that we still feel justified in maintaining our rigid adherence to the rules of Halsted's technic. The mixture of catgut with silk has clinically and experimentally proved unwise in abdominal wound closures.

To illustrate the technic employed, an abdominal opening and closure will be described in brief. An incision of the desired length is made in the right rectus region, with a sharp knife, through the skin only. Then the subcutaneous vessels, arteries and veins are picked up with hemostats. It is important for complete hemostasis that the vessels be clamped as they are met, as the operative field is thereby more readily free of blood. The panniculus carnosus muscle and the areolar tissue down to the fascia of the rectus abdominal muscle are cut, the fascia being exposed by sharp dissection back one-half inch on each side. The entire length of the sheath is incised. The muscle at the upper end in a small area is incised, this being done by pulling the muscle up, then, with a finger or the butt of a knife, separating the muscle the entire length of the cut fascia.

The peritoneum is picked up with a forceps and a clamp at the middle, and incised. All vessels are tied off before the incision is made. The operator clamps off as little adjacent tissue as possible, for good hemostasis. If there is too much devitalized tissue, it is clipped close to the ligature. All the tissues are handled carefully for minimum trauma. The wound is not wiped, but mopped by daubing the desired area. Sponges are placed on the edges of the wound under hemostats that were previously placed in the subdermal tissue, for a complete operative field coverage. In all abdominal operations, silk is used throughout, except for suturing the mucous membranes of the bladder and intestine.

The abdominal wall is closed with four

layers of sutures. The peritoneum is sutured alone with a running, continuous suture knotted at both ends. The second row is an interrupted Lembert suture, the sutures being placed a quarter to a half inch apart. The stitch is made so that the suture goes through the fascia only, thereby involving as little of the nourishing muscle tissue as possible. If the dog is fat, the areolar tissue is sutured at great intervals in order to draw it together with an interrupted stitch. The next layer is an inverted interrupted stitch, started on the inside of the true skin, going into the serosa or true dermis and out again, and over to the opposite side so that the knot is inverted downward away from the outer edge. The last layer is a continuous subcuticular stitch, knotted on each end. This seals the wound.

The writer enucleated an atheroma on a Thoroughbred filly under infiltration local anesthesia by using the technic described above, with first-intention healing as the result. Without complete hemostasis the thick, capsulated cyst could not have been enucleated in its entirety. A year later the scar, which was on the head, was so slight that two horsemen could not locate it.

The extra time required for this systematic, meticulous procedure is offset by shorter post-operative care and better end results. If the dog is hospitalized, only a clinical record is made of the temperature, bowel movement and appearance. The animal is raised by the front legs so that any possible swelling or fluctuation over the abdominal incision can be observed. With this type of closure, no bandage or support is necessary. This is especially advantageous in cesarean sections on succulent nursing bitches.

Ten years ago aseptic surgery was considered impractical for veterinary use. Today it is being done on a fairly large scale, successfully and profitably. This modification in the selection of silk for sutures and ligatures has given good results in all types of abdominal surgery for which we have used it. It is not unusual for us to perform two operations under the one continuous anesthesia.

Those who use silk must be infection-conscious and secure complete hemostasis with minimum trauma. Further, a complete revision of procedure and different instruments are required.

References

[1]Gadd, J. D.: Intratracheal anesthesia. No. Amer. Vet., xx (May 1939).

[2]Halsted, W. S.: Ligature and suture material: The employment of fine silk in preference to catgut and the advantages of transfixion of tissues and vessels in control of hemorrhage; also an account of the introduction of gloves, gutta-percha tissue and silver foil. Jour. Amer. Med. Assn., lx (April 12, 1913), p. 1119.

[3]Shambaugh, P., and Dunphy, J. E.: Postoperative wound infections and the use of silk: An experimental study. Surg., i (March 1937), p. 379.

[4]Kraissl, C. J., Kesten, B. M., and Dimiotti, J. G.: The relation of catgut sensitivity to wound healing. Surg. Gynec. & Obst., lxvi (March 1938), p. 628.

[5]Meleney, F. L.: Infection in clean operative wounds. Surg. Gynec. & Obst., cl (Feb. 1935, No. 2A), p. 264.

[6]Whipple, A. O.: The choice and use of ligature and suture material in the repair of clean wounds. Int. Abst. Surg., p. 109, in Surg. Gynec. & Obst. (Aug. 1939).

[7]The renaissance of silk in surgery. Editorial in Jour. Amer. Med. Assn., cxiii (Oct. 7, 1939).

Cribbing

To the Editor.—Can you refer me to an operation indicated to correct a condition in horses known as windsucking or cribbing?—*S. G. K., Georgia.*

Reply.—Resection of a portion (2 or 3 in.) of the sterno-thyroideus and sterno-maxillaris muscles at the proximal third of the cervical region was once regarded as a cure for cribbing and, 25 to 30 years ago, it was practiced quite extensively. The results, however, were not brilliant. The relief was not permanent. Horses operated upon stopped cribbing only for a short time, a time corresponding, no doubt, to the duration of the soreness produced by the surgical wound.

In later years Forsell practiced a more extensive resection of the same muscles with better results. In effect, the Forsell method amounts to resecting the inferior (anterior) group of cervical muscles over a range of about 15 in. The operation is quite a bloody affair and it leaves a blemishing cicatrix that is objectionable. Regardless of the benefits claimed, the operation never became popular.

Cesarean Section in a Cow

By E. C. JESPERSEN, D.V.M.

Omro, Wis.

THE WRITER was called on December 18, 1939, to see a 3-year-old grade Guernsey cow showing some signs of calving for about one day. Examination revealed a malformed fetus with the limbs flexed and the joints rigid. After spending considerable time in an unsuccessful attempt to amputate the parts, the writer found it necessary to perform a cesarean section.

The operative area, on the left side, was clipped and shaved, then scrubbed and cleansed with etherized liquid soap. The area was dried with cotton and a preoperative disinfectant applied. Should the intestine be full, the opening of the abdominal cavity on the right side may cause considerable difficulty and, for this reason, the left-side operative area is preferred.

Seven per cent chloral hydrate solution intravenously was used to produce anesthesia, which was immediate and complete upon the administration of 500 cc. (1 pt.). The operative area was again painted with a 1:3,000 phe-mer-nite preoperative solution. An incision was made through the skin, starting about 6 in. below the transverse processes of the lumbar vertebrae and midway between the last rib and external angle of the ileum. This incision was extended downward about 15 in. The scalpel used for the skin incision was then placed aside and another scalpel was employed to cut through the external and internal oblique muscles and peritoneum.

The uterus was located and, with some difficulty, lifted toward the flank wound. Sufficient packing was placed about the uterus to prevent the uterine fluid from emptying into the peritoneal cavity. An incision of 10 to 12 in. was then made into the uterus. The fetus was withdrawn and the placenta also was removed, as it was well loosened from the cotyledons. The uterus was stitched with a continuous suture reinforced with interrupted sutures, the serous coats thereby being brought well together. The muscles and peritoneum were stitched together with interrupted sutures, and the skin was sutured separately with interrupted sutures.

The external wound was painted again and the animal was raised on the sternum, propped in that position and blanketed. One hundred cc. (3.3 oz.) of a 50 per cent solution of dextrose was given intravenously.

The operation was performed early in the afternoon and occupied about 1½ hours. The same evening the animal was on its feet and taking food. More dextrose solution was given the next morning, at which time the patient was progressing satisfactorily. At this writing, 22 days following the operation, the cow is well on the way to complete recovery.

The writer prefers to operate rather than to spend hours of trying work in these exceptional cases of dystocia. If the operation is performed carefully, the animal will probably recover.

Colostrum

W. A. H. of Michigan writes: "It is a well-known fact that a cow allowed to go full term will give colostrum for a period of time thereafter. But suppose that the cow is milked a week or two before calving for any reason whatever; would she give true colostrum at the time she was started to be milked or would she give it at the time of calving? In other words, is colostrum the result of an animal's being dry for some time or is it the result of the liberation of hormones by the various internal organs at the time of calving?"

Reply.—If the cow were milked before freshening a week or two, the colostrum (= that milk formed between the dry period and the time that a number of hormones are functioning at capacity after parturition) would be secreted and all

washed out when she freshened, except that there would be a little left in the milk not yet drawn. Some dairymen milk out only a part of the milk before the cow freshens. In this case, more or less colostrum is left and, when the cow freshens and all of the milk is taken from the udder, the remaining colostrum is drawn.

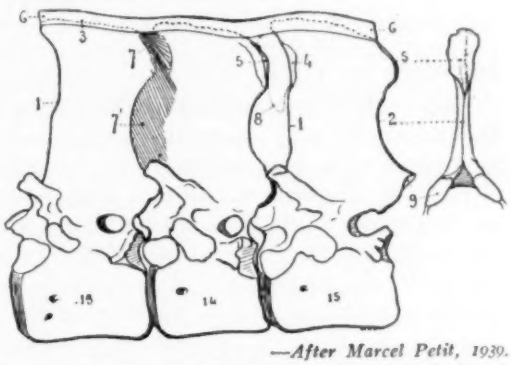
Colostrum, it appears, is formed by desquamated epithelial cells, leucocytes and fat cells which are stored in the udder.—*C. H. Case, V.S., Akron, Ohio.*

Interepispinal Articulations in the Horse

THE EPISPINAL, or more commonly called supraspinous, processes of the dorsal vertebrae may develop veritable articulations between their proximal extremities. Though held intact with the strong supraspinous ligament stretching along the column and interspinous ligaments between each of them, the supraspinous processes are not motionless. They move to and from one another in extension and flexion of the spinal column. This is not without interest from the pathological point of view.

From the first to the tenth segments of the dorsal section of the column, the spaces between these processes, though relatively narrow, do not contact one another. Behind the tenth, the arrangement is different. The tuberosities are flat transversely, with a co-relative antro-posterior development to an extent that they approach one another and play one against the other to such a degree that articulations are formed. Though not found in young animals or in all adults, it was possible at a certain age to find these articulations in practically all subjects examined, particularly between the 13th, 14th and 15th segments. The osteology and arthrology of the region are described in detail, particularly in respect to the thick and strong interspinous ligaments superiorly, where they radiate to form the margins of the articular surface with that of the adjacent process.

The articular cavity is formed by the osseous processes and the interspinous ligaments, extending outward and upward to the supraspinous ligament, where the superior part of the articular cul de sac is formed. Below this the cul de sac varies according to the extent of the articulation. The cavity is lined with a synovial membrane and contains a small amount of liquid resembling synovia.



—*After Marcel Petit, 1939.*

Lateral view (left) of the 13th, 14th and 15th dorsal vertebrae of a horse. 1) Anterior border. 2) Posterior border. 3) Superior border—dotted outlines. 4) Anterior articular surface. 5) Posterior articular surface. 6) Supraspinous ligament. 7) Superior part of the interspinous ligament. 7') Inferior portion of the interspinous ligament. 8) Inferior cul de sac of the articular cavity. 9) Posterior articular process. G = 0.62.

Flexion of the back (arching) separates these apophyses, extension (bending down) approximates them, and lateral incurvation makes them play upon each other. That these articulations can be the site of inflammatory processes in saddle and draft horses can hardly be questioned. Attention to them by clinicians is invoked. (*Marcel Petit. Articulations interépineuses chez le cheval. Revue de Médecine Vétérinaire, xci, June 1939, pp. 314-316.*)

Anaphylaxis is derived from "without protection," and allergy from "altered reaction." Atopy and hyperargy are also used to name this strange phenomenon of protein sensitization.

There were more women physicians in the United States in 1910 than in 1930—9,000 as compared with 6,800. Except that nursing has attracted women in ever-increasing numbers, no reason is given for the decrease.

CURRENT LITERATURE

ABSTRACTS

Selenium

Under the title "Selenium Poisoning," Davidson (W. B.) summarizes the more important facts known about this harmful element of the prairie country. Discovered by Gripsholm (1817) of Sweden, little was known of its toxicology until 1934, when Beath of the Wyoming station showed the relation between plants containing this element and the acute disease of live stock commonly called staggers or alkali disease, together with the danger from wheat raised on land enriched with the organic form of the element by plowing under seleniferous vegetation. T. C. Madison (1856) in Nebraska described the disease among the cavalry horses of the early Indian campaigns, and N. S. Mayo (1891) in Kansas wrote of alkali disease which the early ranchers attributed to drinking water heavily charged with minerals, but which later investigations proved to be erroneous.

The narrow-leaved milk vetch (*Astragalus pectinatus*) and the two-grooved milk vetch (*A. bisulcatus*) are two of the main plants which sap insoluble selenium from the soil and convert it into the soluble, organic form, though many other plants also are capable of doing this to a lesser degree. The danger arises when these "converter plants" impregnate the soil with heavy concentrations of organic selenium. Experimentally the content of wheat was increased from two parts per million to 1,150 parts per million of organic selenium. The danger point is reached in plants containing five parts per million.

Samples of soil collected thoughout Manitoba, Saskatchewan and Alberta show widespread occurrence of selenium in the western part of the Dominion. In certain districts the heavy concentration constitutes a definite menace to live stock. Alberta hay has been found to contain 20 parts per million. In one district samples of *A. pectinatus* were found to contain 2,310 parts per million.

—*Reproduced from Canadian Journal of Comparative Medicine.*

Left: This cow has lost the right front foot, separation having taken place at the fetlock joint. There also has been separation and loss of half of the tail. Center: Astragalus bisulcatus in full bloom (photo taken June 1938). The flower is purple. This cluster is about 18 in. high. Right: Yearling heifer affected with acute selenium poisoning.

The tissues of affected animals autopsied were found by Byers of the U. S. bureau of chemistry and soils to contain small amounts of selenium: Liver, 0.1; spleen, 0.1; and kidneys, 0.2 parts per million. Samples of soil and seleniferous plants from the same areas showed: Soil, 3.5; *A. pectinatus*, 290; and Russian thistle, 4 parts per million, respectively.

The lesions found post mortem were emaciation, erosion of diarthroidal articulations, calcified marrow, fibrosis and atrophy of the liver, hepatic abscesses, petechi-

ation and ecchymosis of the endocardium and other organs, swollen lymph nodes, cardiac atrophy, *et al.*

SYMPTOMS OF SELENIUM POISONING

The chronic form is manifested by a long chain of symptoms summarized as follows: Emaciation, depraved appetite, lameness, loss and loosening of hoofs and hair, gangrene of the extremities (frozen feet), stilty gait, open sores, rough coat, general depression, and anemia. The acute case wanders from the herd, walks blindly and often in circles, grates the teeth, drools from the mouth, eats unnatural material, suffers from colic, presses forward against obstacles encountered, goes completely blind, and dies in a state of paralysis. Calves and lambs of affected mothers may show symptoms of the disease. Lambs born of mothers grazing on seleniferous pastures may be deformed and unable to to control their legs. Toxic feed given to hens causes low hatchability and weak chicks.

TREATMENT

Large drenches of hot water (120 to 130° F.) up to three or four gallons every three or four hours and large doses of strychnine bring a good response if given before the organs are too badly damaged. Chronic cases show some improvement from this treatment when given nontoxic forage. (*W. B. Davidson. Selenium Poisoning. Canadian Journal of Comparative Medicine, iv, January 1940, pp. 19-25.*)

Johne's Disease in Sheep

Though the incidence is not as high as in cattle, the presence of Johne's disease in sheep was recognized and reported from time to time in the past 25 years. Stockman recorded the ovine disease in 1911 and others at rare instances since that date. A classical report by McEwan of an observation commencing in 1932 gives the salient facts which establish this tenacious pseudotuberculous affection of cattle as a threat to sheep.

Clinical cases occurred in the flock in question at all times of the year, but more especially when the ewes were suckling their lambs. Usually, the feces of the infected ewes were heavily contaminated with the specific agent. In the flock of approximately 350 head, 263 were negative to the johnin test: Intrapalpebral test made with johnin derived from the bovine strain. The autopsies showed no constant accord with the postmortem findings, in the reacting ewes.

In sheep the macroscopic lesions may not be sufficient to arouse suspicion, and the microscopic examination of smears may fail to show the presence of acid-fast bacteria, when the gross lesions were typical. . . . Healthy sheep may react to johnin, although where the disease exists, the reactions run higher than among the apparently disease-free flocks. . . . Sheep possess some nonspecific sensitization to johnin and, *per contra*, infected sheep may not react. Johnin of the bovine strain is, therefore, of little value for ovine Johne's disease.

Among the symptoms pointed out are emaciation, cachexia, pultaceous or even watery feces, and pulling out of the wool on handling. The diarrhea is, however, by no means constant. The feces may be formed even in affected animals on succulent pastures. The postmortem gross and histological findings in the intestine and lymph glands are described. . . . Animal inoculation with suspensions loaded with the bacillus were, on the whole, unrevealing in rabbits, guinea pigs and day-old chicks. On the contrary, lambs and calves inoculated with large doses of the suspension developed the disease but the organism resisted cultivation and, in that respect, differs from the organism isolated from cattle.

The annual losses in infected flocks are approximately 5 per cent, but the danger of spreading to other flocks is less than from herd to herd of cattle because the movement of individual sheep from one flock to another is limited. As was shown by Hagan (1938), young animals are the most susceptible. While generally admitted that sheep contract the disease from cattle there are instances where such contact could not be shown. The inability to culture the

organism isolated from sheep appears to indicate the existence of two strains. (*A. D. McEwan. Investigations on Johne's Disease of Sheep. Journal of Comparative Pathology and Therapeutics, lii, March 1939, pp. 69-86.*)

Vitamin A and Louse Resistance

It is commonly observed that domestic animals in poor flesh and poorly fed are frequently heavily infested with lice, particularly in the early spring. This suggested a study of the relation of diet to a resistance to lousiness.

Sixty laboratory rats were infested with the common suctorial lice of rats, *Polyplax spinulosa,* after having been placed on a vitamin A deficient ration and after deficiency symptoms appeared. The lice proceeded to infest all these rats in varying degree, 21 of them becoming heavily infested. Seven out of ten heavily infested rats were able to dispel their lice when given an abundance of vitamin A.

Twelve rats which received an abundance of vitamin A were uniformly free from lice despite several attempts to colonize lice upon them. (*E. M. Searls and F. M. Snyder. A Study of the Relation of Vitamin A to Louse Resistance in Rats. Journal of Parasitology, xxv, 1939, pp. 425-430.*)

Immunity—Canine Hookworms

Three litters of dogs, a total of ten animals, have been used to demonstrate the sharp difference between the active immunity to hookworm resulting from serial light infections and the partial resistance of mature dogs to initial infections.

Dogs were immunized by injecting gradually increasing doses of hookworm larvae subcutaneously over a period of five to seven months at four-day intervals, starting with doses of about 15 larvae and ending with 120,000 to 200,000 larvae. From 52 to 203 adult worms were recovered at necropsy from immunized dogs, whereas the unprotected litter mates produced 1,263 to 31,200 worms. Furthermore, the unprotected animals, with one exception, succumbed to the test infections, whereas the immunized animals were scarcely disturbed.

In the discussion of these results and the previous work on dog hookworm it was concluded that there is nothing distinctive about the so-called age resistance but rather that maturity and general good health are essential for the host to respond fully and quickly to the stimulus of invading worms, *i.e.,* actively to acquire immunity. [*Authors' abstract summary.*] (*G. F. Otto and K. B. Kerr. Immunization of Dogs Against Hookworm, Ancylostoma Caninum, by Subcutaneous Injection of Graded Doses of Living Larvae. American Journal of Hygiene, section D, xxix, 1939, pp. 25-45.*)

Immunity to Ticks

One infestation of guinea pigs or rabbits with larvae of the American dog tick, *Dermacentor variabilis,* induces an acquired immunity which effectively prevents subsequent batches of larvae from engorging. In guinea pigs the immunity develops fully within two weeks after the start of the first infestation and lasts at least three months. Cross immunity to larvae of certain other tick species can be produced.

The immunity of guinea pigs to larvae of *D. variabilis* can be produced artificially by the intracutaneous inoculation of an extract of larval ticks. It can be passively transferred by the intraperitoneal inoculation of serum from guinea pigs hyperimmunized by repeated infestations of nymphs.

In the ears of nonimmune guinea pigs, on the fourth day after attachment of a larva of *D. variabilis,* there is little cellular reaction. In immune animals the mouthparts of the tick are surrounded by a solid mass of leucocytes and the epithelium has thickened and begun to grow beneath the leucocytic mass. In this way, the tick becomes walled off from its source of supply of blood before it can engorge. (*W. Trager. Acquired Immunity to Ticks. Journal of Parasitology, xxv, 1939, pp. 57-81.*)

Correction: In the abstract, "Anesthesia of Foxes and Mink," published in the February 1940 issue, page 277, the words "sodium sulfate," lines 4 and 5 and 8 and 9 of the fourth paragraph, should have read "sodium sulfide."

Staphylococcic Anatoxin in the Treatment of Dermostaphylococcic and Nonstaphylococcic Dermatites in Dogs

Forty cases of staphylococcic infection of the skin were treated with anatoxin. The cases were either true staphylococcic infection or associated with dorso-lumbar eczema or demodectic mange. The diagnosis, when possible, was confirmed by cultural methods —inoculation of gelose with scrapings. Regardless of the form, improvement followed the fourth to the sixth injection. The congestive and inflammatory phenomena of the skin declined rapidly; purulent discharges streaked with blood became fluid, then hemorrhagic, rosy and serous. In certain cases the pustules were slow in drying up and a yellow, viscous secretion that agglutinated the hair persisted. This was particularly the case in the interdigital lesion.

Relapse in lesions apparently cured occasionally occurred, but these are not important if the treatment is continued. Strangely, older cases respond better than recent cases in young, robust dogs. Individual behavior of the patients does not appear to be influenced by age, sex, age of the lesion, or extent of the invasion. It was observed that the receptivity of dogs to staphylococcic infections is influenced by distemper and infectious pneumonia.

Many cases develop during the days or weeks following convalescence from these diseases. Dermostaphylococcia of a somewhat stubborn nature often follows preventive vaccination against distemper. When the infection occurs in a young dog that has not had distemper, it is relatively benign and rarely generalizes. When distemper breaks out, the dermatitis attenuates or completely disappears, to reappear, however, with increased intensity after recovery or during convalescence.

In regard to the posology of staphylococcic anatoxin, the recommendations of Holstein and Richou (*Bulletin de l'Academie de Médecine Vétérinaire de France*, March 1938) were followed at first, but since certain animals reacted strongly to the initial injections, the doses were diminished to ¼, ½, and ¾ cc. With these doses the treatment was tolerated much better. These first series of doses apply to all dogs, regardless of size or breed. For the subsequent doses, a Scottish Terrier of 6 months supported 1½ to 2 cc. after the fifth or sixth dose, as did a German Shepherd of 5 years.

Seventeen cases of dermostaphylococcia are described in detail as to the extent and intensity of the cutaneous involvement and the results obtained. These, in general, were remarkable. Prompted by this success, the author thought it would be interesting to determine the value of staphylococcic anatoxin in various types of nonstaphylococcic dermatites (eczemas). Twenty-five such cases were treated with the following results:

1) The pruritus, the erythema, the vesication and the oozing were but little modified by an average of six to eight injections.

2) The impetigo was always favorably modified. Certain infected eczematous patches dried up after two to four injections.

3) The lichenization and hyperkeratosis were always attenuated and often entirely cured when the treatment was continued for a sufficient period of time. For example, lichenoid patches on the belly, flank and fetlock disappeared completely after ten to twelve injections. The hyperkeratosis of truffle eczema was not modified.

4) The small, torpid ulceration on the margin of the natural orifices and certain buccal exulcerations which accompany various tenacious, recurrent eczemas were strikingly ameliorated and often cured.

5) Simple or suppurative otitis externus was often radically cured and always favorably influenced.

The author was convinced that, in general, dogs affected with infected eczema obtain great benefit from staphylococcic anatoxin. The results registered appear to

be much better than those from any other medication, general or local. (*Jean Buzenac. Observations cliniques concernant le traitement des staphylococcies cutenées du chien par l'anatoxine staphylococcique. Recueil de Médecine Vétérinaire, cxv, September 1939, pp. 531-539.*)

Effect of Heat on Trichina Larvae

Infected rats were subjected to artificial fever by means of diathermy in an effort to destroy trichina larvae. The treatment did not injure the larvae although five of the six rats used succumbed to the effects of overheating.

Heating larvae in glass tubes confirmed earlier researches showing that 55° C. is the lowest temperature at which trichina larvae are quickly killed. This affirms that the federal requirement of 137° F. (= 58.3° C.) in the heat processing of pork is adequate in destroying trichina larvae. (*G. F. Otto and E. Abrams. Quantitative Studies on the Effect of Heat on Trichina Larvae. American Journal of Hygiene, section D, xxix, 1939, pp. 115-120.*)

BOOK NOTICES

Angiology and Splanchnology of the Horse

R. L. Mundhenk, professor of anatomy and histology at the Alabama Polytechnic Institute, publishes a spiral-bound brochure in modernistic offset printing that is a companion of his treatise on osteology, arthrology and myology. It is a dissecting laboratory manual containing explicit directions on the preparation of a dissecting subject and the component parts of the apparatus indicated in the title.

The author interpolates original ideas on the teaching of anatomy which depart somewhat from the orthodoxy of the art. Frequent inspection of a living horse kept in the laboratory for the purpose of locating on the living body the structure being dissected is pointed out as an important step in the teaching of structural organization. The student is advised against making the dissecting laboratory a study room. Anatomy should be studied at home with the living body kept uppermost in mind. Memorizing should be avoided in the study of blood vessels; the related bones and muscles must be kept in mind as the study of angiology proceeds.

The study of the pulse and murmurs of the lungs on the living subject about to be killed is another detour from the conventions, as are also the multiplicity of details recommended for the handling of all of the branches of the circulatory tree, and abdominal organs. As a manual on the technics of dissecting the equine cadaver, this booklet seems to leave little to the imagination. (*Angiology and Splanchnology of the Horse. By R. L. Mundhenk, professor of anatomy and histology, Alabama Polytechnic Institute. Edwards Brothers, Inc., Ann Arbor, Mich., 1939. 98 pages. Price, $1.75. Orders should be placed directly with the author.*)

Milk's Fourth Edition

The fourth edition of the book we know best as "Milk's Materia Medica" has just appeared in a truly revised and enlarged form.

A chapter on biological therapeutics by Adolph Eichhorn has been revised, and sections on the autonomic nervous system and sex hormones have been added. The section on vitamins is brought up to date and many new drugs, not contained in previous editions, are described. The long list of these includes such popular newcomers as sulfapyridine, prontosil, halital, lentin, colloidal sulfur, thiamin, nicotinic acid, riboflavin and salyrgan, to name but a few. The remarkable progress made in chemotherapy and biological therapy during recent months is well indicated by these revisions and additions. So rapidly have new remedies and new conceptions of old ones come upon the practice of medicine that authors of books on this subject are obligated to revise, add and delete *ad eundem* in order to keep in step with the march of science.

Reading successively the prefaces of the four editions without comparing the editions themselves gives quite a true image

of the pharmacological developments of recent years. The text is approximately the same as that of the third edition, editing is excellent, typographic errors rare, paper good, and binding esthetic.

The evaluation of some of the new drugs is open to argument, but pharmacodynamics were ever thus, awaiting the consensus established by time and experience. The section on vitamins seems too brief, although with an exception or two none of the main facts is slighted.

The chapter on biological therapy is fine reading, clear, edifying, concise. Present-hour conceptions of the main biological products used in the practice of veterinary medicine are well told. For canine distemper the Laidlaw-Dunkin method is preferred.

The variant opinions on the immunizing value of rabies vaccine are mentioned and the variable concentrations of the virus in the brain tissue constituting the vaccine product are given as a possible cause of the controversies. The cities in the United States where excellent results have been obtained through compulsory rabies vaccination are named for those who are seekings facts established by usage. Written by Eichhorn, whose wealth of knowledge in animal pathology and biological therapeutics belongs in the upper cadre, the statements relative to this important branch of veterinary practice are not questioned.

As to Milks, he has been at the job of writing materia medicas for a long stretch of time. He ought to know, and does. He has never tricked his audience with wild theories, and one takes what he says as a matter of course. *(Practical Veterinary Pharmacology, Materia Medica and Therapeutics. Fourth edition. By Howard Jay Milks, D.V.M., professor of therapeutics and director of the small animal clinic, New York State Veterinary College, with a chapter on biological therapeutics by Adolph Eichhorn, director of the Animal Disease Station, U. S. bureau of animal industry. Alexander Eger, Chicago, 1940. 620 pages. Price, $6.00.)*

Fleas of Eastern United States

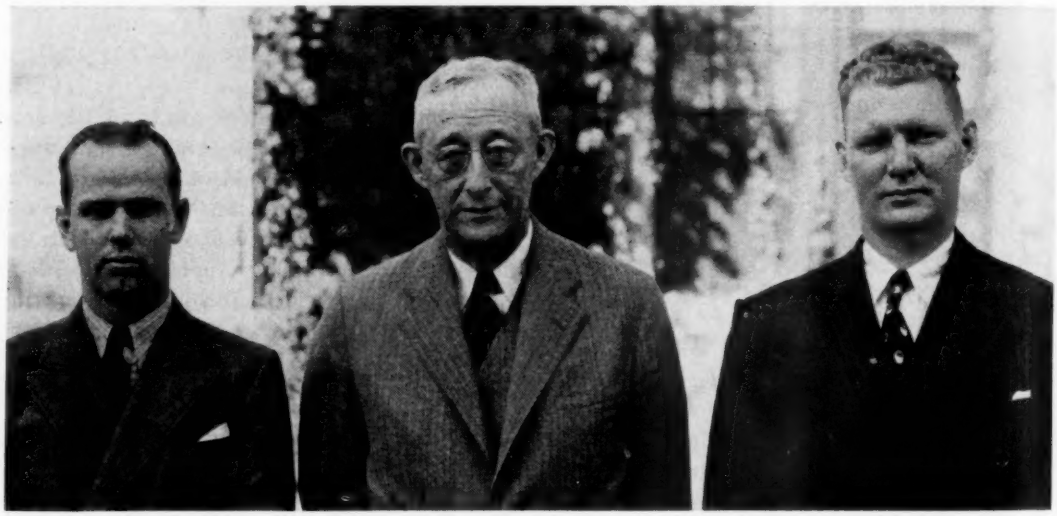
From Iowa State College comes a book reminding one of the growing importance of fleas in the study of medicine. From practically no attention paid to fleas up to the turn of the century, the study of fleas by the entomologist is now being invoked to a remarkable degree. Sketchy papers on the subject are being replaced by classical books containing the known facts about the various orders, families and species which inhabit various parts of the country and parasitize the various species of animals. The rôle of Siphonaptera in the transmission of disease and, as veterinarians well know, the distress and local reactions it is capable of causing have brought this wingless insect into the limelight in the practice of medicine.

The book at hand is the study of the flea *per se*, a purely scientific volume that does not wander into the field of pathology, but gives the reader directions in an introductory chapter on how to capture and study fleas—their morphology, nomenclature, life history and control. Beyond this is the modern classification of the fleas infesting man and the domestic animals: Dogs, cats, hogs and chickens. By treating the body of the infested and destroying the breeding places, the human and domestic animal fleas can be eliminated. In wild life and in rats, on the contrary, the animal itself must be caught and killed.

The body of the book containing the names, zoölogical classification, morphology and geographic distribution, we leave to the connoisseur of entomology to appraise. On the other hand, the 21 plates appended to clarify the text are manifestly informative even to the novice. After studying them one knows just what a flea really is and how it succeeds in making such a nuisance of itself. So, if interested in fleas, and what veterinarian isn't, here is a book which deserves attention. *(Fleas of Eastern United States. Irving Fox, department of zoölogy and entomology, Iowa State College. Iowa State College Press, 1940. 180 pages. Price, $3.00.)*

Poultry Practice

Coming at a moment when the plea for more general attention to the diseases of poultry by practitioners is being heeded on an increasing scale, *Poultry Practice* is a timely book. Announced as "A Collection of Discussions on Poultry Diseases and Related Subjects," reprinted from articles published in *Veterinary Medicine*, the text will be found to exceed all expectations. Edited by an authority in that branch of veterinary science, Professor Leland D. Bushnell, with the aid of his coworkers at Kansas State College, the effort to give the practitioner a liberal education on the everyday problems of clinical avian medicine has been well fulfilled.



—*Cut courtesy Veterinary Magazine Corporation.*

The manuscript contributions which comprise "Poultry Practice" were assembled by Leland D. Bushnell (center) in collaboration with Marvin J. Twiehaus (left) and E. J. Frick (right), all of Kansas State College.

Thirty-four major subjects are covered by capable authors who succeed in bringing poultry pathology, sanitation and therapeutics up to date in concise form without notable omission. The names of the authors are revealing: Martin; McLeod; Thompson; Graham and Hester; Brandly; Beach (J. R.); Feldman, Beaudette; Stubbs; Lumb; Card; Emmel; Van Roekel, Bullis and Clarke; Bushnell, Jungherr and West (L. L.); Bunyea and Zumbro; Carpenter (C. D.); Twiehaus; Frick; Durant; Ackert; Davis; Stafseth; Wehr; Shillinger and Coburn and Edmunds. A glance at the names of these authors is impressive, to say the least, and reading what they have to say gives one a quick insight into the fundamentals of poultry medicine.

Particularly *au fait* is "The Challenge of Poultry Mortality to the Veterinary Profession," by J. Holmes Martin, director of the Regional Poultry Research Laboratory of East Lansing. "Here it is, it is up to you, Doctor," is how this prominent figure of that realm throws the poultryman's hat into the ring. Throughout, the question of the high mortality which harrasses poultry production is kept in the foreground and emphasized as one requiring the replacement of quackery by classical application of medical science. Here, as in so many other branches of medicine, science far exceeds its rational use.

The chapters on diagnostics by Fitch and Pomeroy and by Twiehaus lay down in a few pages a foundation for poultry-disease work that clarifies the whole subject in masterly fashion. Peterson and Thompson

outline the known facts about mineral and vitamin therapy which "hold water." The story of leucosis, moot problem of poultry husbandry, is well told by Stubbs. And who is not always impressed by the writings of J. R. Beach on poultry subjects, of A. J. Durant on blackhead of turkeys, of Beaudette on respiratory infections, of Stafseth or Emmel on poultry parasites, not to mention the other authors, named above, who have thus volunteered to give the practitioners a valuable treatise on a somewhat neglected branch of farm-animal medicine which, as far as the subjects covered is concerned, neither burdens the mind with surplus details nor omits any salient points of interest.

The publisher is to be congratulated for clothing these valuable texts with a solid binding in lieu of the pamphlet type of cover suggesting but transient value. *Poultry Practice* is a book to read through from cover to cover as a means of acquiring a working knowledge of the main diseases of farm poultry and then to place in the library as a convenient reference on what was known about poultry diseases in 1940. *(Poultry Practice. By Leland D. Bushnell, B.S., M.S., Ph.D.; Edwin J. Frick, D.V.M.; and Marvin J. Twiehaus, and others. Veterinary Magazine Corporation, 7632 S. Crandon Ave., Chicago, 1940. 160 pages, illustrated. Price, $1.00.)*

Biological Products

Biological Products is a discourse covering the subject indicated by the title as far as human medicine is concerned. It dwells upon the antitoxins, serums, bacterins, vaccines, toxoids, antigens, and the nonspecific proteins used in the practice of medicine. In the chapter on rabies vaccination, the veterinary side of the subject is presented at some length.

Physical properties, history, detailed methods of production, approved strengths, control, biological assays and standardization, dosage, dynamics, harmful sequels, prophylactic and curative values, and indications are the outstanding captions declaring the author's intentions. Each of these contains a wealth of informative details for the student, producer and user, and as far as can be determined by a casual review of the material, but few of the basic facts are omitted.

The author questions the curative properties of tetanus antitoxin but, nevertheless, gives explicit directions for its use in that rôle, and, in effect, the same is said of gas-gangrene antitoxin (*Antitoxinum welchi*). In regard to staphylococcus antitoxin, "its use will never become widespread until a more potent therapeutic antitoxin or serum is made available," the author declares. All of the antiserums in use are separately evaluated, though the author points out that the U. S. Public Health Service requires official testing of but three: Antipneumococcic, antimeningococcic and antidysenteric. In the discussion on antianthrax serum, the well-known work of Eichhorn in the development of a spore vaccine and its extensive use in human medicine is mentioned. Data show that it decreases the mortality by two thirds. Here, a prominent figure in the field of veterinary medicine is given due credit for his brilliant work.

The polyvalent antistreptococcic serums are not praised. Their action is erratic and uncertain. The trend is toward the production of a specific antistreptococcic serum made by immunizing animals against definite strains of streptococci. In regard to the other antimicrobic serums on the market, the author expresses hope that the future will bring forth improvements in serum therapy, in view of the constant experimentations that are being carried out.

Contrary to established (official) usage in veterinary medicine, vaccines are defined as either killed of attenuated infective agents —capable of producing active immunity but not the disease. They may be employed for prophylactic or curative purposes. Methods of preparation are described. Many pages are devoted to typhoid and typhoid-paratyphoid vaccination, and to the biological treatment of such important maladies as diphtheria, whooping cough, cholera, and plague.

For the use of tetanus toxoid (alum precipitated), two 5-cc. (.17-oz.) doses at an interval of three or more months are recommended. The immunity is established

after a further interval of three to six months. Immunized persons need not be treated with antitoxin when wounded, though giving an additional dose of toxoid is advisable in lieu thereof, when treating the wounded subject. The extensive observations of Ramon and his coworkers on the military horses of France, which furnished the first authenticated facts on the immunizing properties of tetanus toxoid on a large scale, are not mentioned, though Ramon is quoted as recommending the simultaneous use of antitoxin and toxoid as the means of producing a high degree of immunity.

Tuberculin, mallein and johnin as used in veterinary medicine are mentioned but, naturally, not in revealing details. The various tuberculin tests employed by physicians (Mantoux's, Von Pirquet's, Moro's, Callmette's, subcutaneous) are, however, fascinating studies in diagnostics.

The chapter on antivenom serums is of interest in human and veterinary medicine alike. The twelve pages devoted to this subject contain the boiled-down facts on the venom of various snakes and insects: Snakes, spiders, scorpions, bees. Methods of collecting the venom, the preparation of antivenin and the treatment of the victims of these toxic accidents are given. "Just as it is necessary to capture a dog when bitten so as to find out if the animal has rabies, so one should attempt to find out whether the snake that did the biting is poisonous or nonpoisonous," is capital advice. In snake or insect bites, identifying the biter is imperative in the selection of the right antivenin.

Treating the wound promptly with fuming nitric acid and keeping the dog under supervision to determine whether it has rabies are given as the preliminary steps *par excellence* in the handling of a dog bite. Methods of producing a *virus fixé* and of attenuating it in the preparation of the vaccine of Pasteur, Hogyes, Semple, Fermi, Harris, Cunningham, Phillips and Cummings are pointed out.

The comparative freedom of dogs and their biting habit is given as the reason for the prevalence of the disease in that animal. The incubation period of a street-virus infection (dog bite) runs from 15 days to three months. An injection of fixed virus causes the disease in seven or eight days.

Of vaccination of dogs against rabies, the author says: "Though veterinary biological products are not considered in this volume, it is deemed advisable briefly to consider this preparation, inasmuch as methods of preventing rabies in dogs will not only save these animals but will aid considerably in reducing the incidence of hydrophobia in man."

The phenol-killed vaccine should contain 20 per cent of brain and spinal cord tissue suspension, and chloroform-killed 33⅓ per cent. A dose of 5 cc. (.17 oz.) subcutaneously is required for small dogs, and 10 cc. (.33 oz.) for larger ones. The dose should be repeated once a year. Animals bitten by rabid dogs should be given five daily injections and kept isolated for six weeks. The work of Webster and Clow is briefly reviewed. For these authors the production of a tissue vaccine in lieu of the brain-tissue suspension seems desirable, since the brain tissue is not only superfluous but potentially dangerous. Webster's mouse test for measuring the potency of rabies virus is mentioned but not described (see JOUR. A.V.M.A., xcv, January 1940, pp. 65-73).

The closing paragraph, entitled "Veterinary Biologicals," names the groups of animal diseases in which biological therapy is employed and refers the reader to the JOURNAL of June 1930 and the *Proceedings of the 11th International Veterinary Congress* (London) for details.

Except for pictures of laboratory rooms and equipment, the book is but sparsely illustrated. (*Biological Products. By Louis Gershenfeld, P.D., B. Sc., Ph. M., professor of bacteriology and hygiene, Philadelphia College of Pharmacy and Science. Romaine Pierson Publishers, Inc., New York, 1939. 242 pages, illustrated. Price, $4.00.*)

U. S. currency (paper) is quietly being picked up by wealthy Europeans as the best bet against the depreciation of the money of their own countries. Bills of denominations from $1,000 up are reported to be in great demand.

THE NEWS

A.V.M.A. Activities

President Way has appointed J. G. Hardenbergh of Plainsboro, N. J., as chairman of the Committee on Resolutions to succeed the late, lamented C. P. Fitch.

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The Veterinary Association of Manitoba is now a component of the A.V.M.A. through an action taken at its 50th annual meeting, held at the Marlborough Hotel, Winnipeg, on February 9, 1940. R. H. Lay of Winnipeg was elected delegate to the House of Representatives, and H. H. Ross of Brandon, alternate. Manitoba thus becomes the third Canadian province to affiliate; British Columbia was the first (see November 1939 JOURNAL, p. 653) and Ontario the second (see January 1940 issue, p. 128).

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The election of a member of the Executive Board for the third district (Illinois, Indiana and Wisconsin) will begin March 4. The member elected will take office at the close of the Washington meeting. Herbert Lothe of Waukesha, Wis., now represents the district.

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Plans are now being made for holding an extraordinary session of the Women's Auxiliary at the Washington meeting, at which time a schedule of reorganization will be presented for approval.

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The Board of Governors (Jakeman, Way, Wight) met at the Mayflower Hotel in the national capital on February 10 to confer with the local Committee on Arrangements in regard to the organization of the coming convention. President Way was one of the principal speakers at the annual meeting of the Illinois State Veterinary Medical Association in Springfield, Ill., February 15-16, and at the dedication ceremonies of the new veterinary building at Colorado State College, Fort Collins, on February 20. (See Dr. Way's Illinois address, p. 377 of this issue.)

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Executive Secretary Merillat and D. M. Campbell of the Committee on Public Relations will attend the veterinary conference at Alabama Polytechnic Institute in Auburn, March 20-23, which will be held in conjunction with the annual meeting of the Alabama state association.

Assistant Executive Secretary Ingmand will participate in the program of the Arkansas Veterinary Medical Association at Hot Springs, March 21-22.

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The returns from the vote required to legalize the transfer of the Ohio River relief fund of 1937 to the general relief fund show that the contributors practically all approve the project.

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Abstracts of publications on parasitology are now being prepared for the JOURNAL by Associate Editor Benbrook. E. E. Hamann of East Lansing, Mich., and A. G. Karlson of the University of Minnesota, St. Paul, Minn., have volunteered their services in abstracting the German and Scandinavian literature, respectively.

"What the Veterinary Profession Means to Mankind"

Allied Laboratories, Inc., inaugurates with this issue a new policy of advertising to the veterinary profession. With the core idea of portraying how veterinary accomplishments of the past have brought the profession to its present status and have aided world progress, the organization presents as its first chapter, "The Building of the Panama Canal." (See page iii.)

The executives of Allied Laboratories have announced their intention to continue this series indefinitely, offering in each issue a reflection on some outstanding accomplishment of veterinary science.

Veterinary Congress on Artificial Breeding to Be Held in Italy, May 25-26, 1940

Upon the suggestion of the Italian Experimental Institute "Lazzara Spallanzani" for the Artificial Breeding of Animals, of Milan, and the Fascist National Syndicate of Veterinarians, the Second National Congress of Veterinarians for the Artificial Breeding of Animals will convene at Foggia (Apulia) on May 25-26, 1940.

The general secretaryship is assigned to the Institute "Lazzara Spallanzani" and to its director, Professor T. Bonnadonna.

STUDENT CHAPTER ACTIVITIES

Colorado State College

At a meeting held on January 8, the following men were elected to direct the Colorado chapter for the ensuing year: Jack K. Bushnell, president; Jack C. Fletcher, president-elect; Owen Andrus, vice-president; Harris Sorensen, secretary; Vincil Bishop, treasurer; and Merle Dawson, sergeant-at-arms. R. W. Davis of the faculty was elected sponsor.

The chapter is now preparing a bulletin comprising articles and case reports written by members of the senior class. Alumni news and a story on the dedication of the College's new veterinary building also will be included in the publication.

Cornell University

In May 1939, the members of the class of 1940 of the New York State Veterinary College at Cornell University met to discuss the reëstablishment of a student chapter. The proposal met with unanimous approval, and James A. Baker, Ralph Loomis, Ralph E. Witter, Carleton Kelsey and Emery Wingerter were elected president, vice-president, secretary, treasurer and member-at-large, respectively.

During the summer a constitution was drawn up by the chapter's executive committee and, at the first regular meeting in September, this document was voted upon and accepted. The actual activities of the chapter began at that time. A spirited membership campaign was conducted throughout the College, with the result that every student—a total of 163—joined the chapter. Fifty per cent of the present membership subscribe to the JOURNAL, and 75 per cent of the seniors are wearing the special student key.

Once each month there is held a brief business meeting followed by a literary program or some type of entertainment. A banquet and dance are being planned for the May meeting.

At the December gathering, officers for 1940 were elected as follows: Edgar W. Tucker, president; Delano L. Proctor, Jr., vice-president; Miss Jean Mackerly, secretary; Edward F. Steinfeldt, treasurer; and Morton Meisels, member-at-large.

Iowa State College

During the first half of the college year, Frank Connor served as president of the Iowa chapter; Robert Fisher, vice-president; John Carey, secretary; and Arthur Gathman, treasurer.

A complete report of the chapter's activities thus far in 1940 will appear in one of the coming issues.

Kansas State College

At a meeting held on January 16, the Kansas chapter of the Association elected Ted Beard as president; Bob Lank, vice-president; Clifford Lemen, secretary; Carter Anthony, treasurer; Ralph Knocke, marshall; and Bob Erickson, critic.

On February 6, J. S. Hughes, professor of biochemistry at the College, addressed the members on the subject, "The Effects of Chemical Compounds on the Living Cell."

Michigan State College

The Michigan State College student organization succeeded in obtaining a number of the veterinary profession's best-informed speakers for the literary sections of its 1939 meetings. Among the personalities and subject contributions standing out in the chapter's various programs are Lacroix of Illinois on small animal medicine, Stafseth of Michigan on veterinary practice in Norway, Ebright of Indiana on cat practice, and Sweebe of Illinois on small animal anesthesia.

At an election held on May 10, 1939, Gaylord Hartsough was voted president; Antoon Busser, vice-president; Miss Norma Greiner, secretary; and James Schieve, treasurer.

Ohio State University

Lectures on accounting and business management are a new departure in the organization of Ohio chapter programs and, judging from the enthusiastic way that the members are receiving them, will undoubtedly be drafted as an integral part of every meeting that they are available.

Melvin J. Hatter is president of the chapter; Wm. Piper, vice-president; Roy Davison, secretary; and Lawrence Price, treasurer.

Ontario Veterinary College

Guided by the time-proved Dominion technic, students of the Ontario Veterinary College have given careful deliberation and sincere effort to build the well-organized chapter which they now have. Although not all of the students are as yet participating in the activities of the group, it seems improbable that this excellent organization will not attract the support of the entire student body.

K. F. Burns is president; F. L. Clark, vice-president; and J. J. Fanning, secretary-treasurer.

Texas A. & M. College

The Texas chapter has accepted the charter and model constitution designed by the central

U. S. GOVERNMENT

Production of Bang's Disease Antigen

One of the largest laboratory projects undertaken in recent years is the production of Bang's disease antigen in sufficient amounts to carry out the official testing program.

To produce 60,000,000 cc. of tube antigen and 360,000 cc. of plate antigen, which are the estimated annual requirements of the various states, required extensive expansion of buildings and equipment. The investigation leading to this tremendous project shows that the test antigens in the various commercial laboratories lacked the necessary uniformity of strain, culture mediums, and methods of determining density of the finished product. The result was a wide difference in sensitivity and, therefore, in diagnostic uniformity. As this mass production is for official use only, state and private laboratories are furnished with the information required to standardize all test antigens used in Bang's disease eradication.

The Coming Census

The periodicals service section of the Bureau of Census, Department of Commerce, is coöperating with the JOURNAL'S plan of taking a census of the veterinary profession, about to be launched from the central office of the Association. Veterinarians are requested to give careful attention to the questions asked in order that an accurate picture of the veterinary service of the United States may be obtained.

The Association's program is to distribute a printed questionnaire with a request that it be filled out promptly and returned in a prepaid cover. The members of the Association will be canvassed first, then the non-members and, finally, the non-graduates.

The government's general census will be taken in April, when 120,000 enumerators will visit 32 million families and gather data on the nation's estimated population of 132 million. This will be the sixteenth decennial census. The first one was taken in 1790, in compliance with article 1, section 2 of the federal constitution. The population at that time was 4 million and it required a year and a half to complete the job. By means of a huge battery of mechanical tabulators the census of 1940 will come out almost as fast as the enumerators take it.

(Continued from preceding page)

office of the Association and, according to a recent report of Secretary-Treasurer Vernon Isaac, the organization is progressing in a "better than ever" style. Other officers are R. J. Rodgers, president, and Ole H. Stalheim, vice-president. J. R. Ketchersid and J. H. Milliff of the faculty are the sponsors.

Sixth Corps Area Will Hold Training Course for Medical Department Officers

The third annual military medico-dental training course at Chicago for medical department reserve officers will be held by the sixth corps area during the period March 31 to April 13, 1940.

This course, which will be conducted under the direction of the corps area surgeon, Colonel Paul W. Gibson, medical corps, with the coöperation of the medical and dental schools of the University of Illinois, University of Chicago, Loyola University, and Northwestern University, is designed to increase the military and professional proficiency of reserve officers of the medical, dental, veterinary and medical administrative corps. The morning hours will be devoted to professional work in the schools, hospitals and clinics connected with the participating universities. The afternoon and special evening sessions will be devoted to problems connected with the operation and functioning of the medical service in war. The military courses for the current year will be especially concerned with problems of mobilization as they affect the medical department of the army.

Previous courses of this type, conducted in Chicago in 1938 and 1939, met with an enthusiastic response. Members of the veterinary profession who are officers of the veterinary reserve corps and interested in attending this course should communicate with the Surgeon, Sixth Corps Area, Room 1040, U. S. Post Office Bldg., Chicago, Ill.

Regular Army

Lt. Colonel Forest L. Holycross is relieved from additional duty as attending veterinarian at Maxwell Field, Ala.

By direction of the president, and under the provisions of Public, No. 18, 76th Congress, 1st Lieut. Harold Myers Deane, veterinary corps reserve, is ordered to active duty effective January 15, 1940, and assigned to duty at headquarters, Ninth Corps Area, Presidio of San Francisco, Calif., until February 1, 1940, when he will proceed to Seattle, Wash., and report to the commanding officer, Seattle quartermaster depot, for duty.

Lt. Colonel Peter T. Carpenter, Fort Logan,

Colo., is relieved from additional duty as attending veterinarian, Lowry Field, Colo.

Each of the following-named officers of the veterinary corps is relieved from his present assignment and duty at the station indicated after his name, effective in time to proceed to New York, N. Y., and sail on transport scheduled to leave that port on or about April 2, 1940, for the Philippine department and, upon arrival, will report to the commanding general for assignment to duty: Lt. Colonel Mott Ramsey, Jefferson Barracks, Mo.; and Captain Albert A. Roby, Jr., Fort Hoyle, Md.

Lt. Colonel Harry E. Van Tuyl is assigned to duty at Fort Sheridan, Ill., effective upon completion of his present tour of foreign service in the Philippine department.

Lt. Colonel Fred C. Waters is assigned to duty at the Chicago quartermaster depot, Chicago, Ill., and to additional duty at headquarters, sixth corps area, effective upon completion of his present tour of foreign service in the Philippine department.

Veterinary Corps Reserve

NEW ACCEPTANCES—FIRST LIEUTENANTS

Samuel Edward Grove, 1101 Park St., Fort Worth, Texas.

James Howard Hathaway, 2307 Ellis Ave., Fort Worth, Texas.

Chas. Calvin Harman, Jr., Greenville, Ga.

Samuel Jack Levine, Sidney Road, Waterville, Me.

Elmer Lavern Matthews, Route No. 3, Milton, Fla.

Phillip Woodrow Tedder, Edenton, N. Car.

Edwin Joseph Sunderville, Forest Home, Ithaca, N. Y.

PROMOTION—TO CAPTAIN

Harold Loran Morrison, Gilmore City, Iowa.

B. A. I. Transfers

Joseph H. Adams from Charleston, W. Va., to Harrisburg, Pa., on Bang's disease.

Oscar W. Anderson from South St. Paul, Minn., to Fort Dodge, Iowa, on meat inspection.

Benjamin Z. Burleson from Atlanta, Ga., to Montgomery, Ala., on Bang's disease.

David H. Carter from Columbia, S. Car., to Montgomery, Ala., on Bang's disease.

Albert V. Dixon from Ottumwa, Iowa, to Raleigh, N. Car., on Bang's disease.

Salem G. Fine from Richmond, Va., to Augusta, Me., on Bang's disease.

Silas J. Goldstein from South St. Paul, Minn., to Milwaukee, Wis., on meat inspection.

Kenneth W. Irvin from Madison, Wis., to St. Paul, Minn., on Bang's disease.

George S. Jones from Columbia, S. Car., to Montgomery, Ala., on Bang's disease.

Robert P. Lusco from St. Louis, Mo., to Knoxville, Tenn., on meat inspection.

Gerald H. McChesney from Madison, Wis., to Atlanta, Ga., on Bang's disease.

Clarence H. Pals from Fort Dodge, Iowa, to Sioux City, Iowa, on meat inspection.

John Redmond from Jacksonville, Fla., to San Antonio, Texas, on tuberculosis eradication.

Raymond R. Rohrer from Raleigh, N. Car., to Ottumwa, Iowa, on meat inspection.

Daniel B. Schlosser from Charleston, W. Va., to Harrisburg, Pa., on Bang's disease.

Morris D. Schneider from Madison, Wis., to St. Paul, Minn., on Bang's disease.

Ralph E. Springer from Columbus, Ohio, to Jefferson City, Mo., on field inspection.

Samuel Stieber from Raleigh, N. Car., to South St. Paul, Minn., on meat inspection.

Benjamin H. Yenner from Knoxville, Tenn., to Evansville, Ind., on meat inspection.

Moses R. Zinober from Madison, Wis., to St. Paul, Minn., on Bang's disease.

Retirements.—Clarence Upton, Evansville, Ind.; James H. Cock, Omaha, Neb.; Peter I. Kershner, Topeka, Kan.; Carl M. McCoy, Oklahoma City, Okla.; Oscar Nelson, Jersey City, N. J.; Charles E. Windeler, Chicago, Ill.

AMONG THE STATES

Arizona

Karl F. Meyer of the George Williams Hooper Foundation, University of California, San Francisco, Calif., will speak in behalf of veterinary interests at a meeting of the Arizona Public Health Association to be held in Tucson, April 16-17. F. L. Schneider of Albuquerque, N. Mex., and Myron Thom of Pasadena, Calif., will contribute to the veterinary section of the convention.

Connecticut

The annual meeting of the Connecticut Veterinary Medical Association was held in Hartford at the Hotel Bond on February 7.

Officers for the ensuing year were elected as follows: F. I. Maxon of Hartford, president; J. M. Curry of Hartford, first vice-president; Niel W. Pieper of Middletown, second vice-president; and Geo. E. Corwin of Hartford, secretary-treasurer (reëlected). F. F. Bushnell of Manchester, G. L. Cheney of Woodbridge, G. H. Ludins of Hartford, J. J. Kavanek of Hartford, and E. M. Bitgood of Middletown were elected to the Board of Censors.

T. W. Workman, state deputy dairy and food commissioner, was unanimously elected an honorary member of the Association.

A. Gordon Danks of the New York State Veterinary College at Cornell University, Ithaca, N. Y., spoke on "New Treatments and Surgical Technics." His talk was well received and it was generally agreed that this was one

of the most practical presentations ever featured on a Connecticut state program.

GEO. E. CORWIN, *Secretary.*

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Arrangements are being made by the state department of domestic animals to test goats for tuberculosis and Bang's disease. An appropriation of $1,000 has been made and the program will be carried on in a manner similar to that of testing cattle for these diseases. The project is intended to help owners of goat dairies to comply with new regulations set up by the state milk regulation board, which requires that all goat milk sold for consumption must come from herds officially tested for both tuberculosis and Bang's disease. A number of tests already have been made and the results to date show that few, if any, goats in Connecticut are affected with either of the diseases.

Indiana

The Northwestern Indiana Veterinary Medical Association held a small animal clinic at the hospital of Charles Gruber in Fort Wayne on February 13. J. A. Campbell of Toronto, Ont., was the chief clinician and local veterinarians assisted him. The clinic was followed by a banquet at the Catholic community center.

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The 52nd annual convention of the Indiana Veterinary Medical Association, with 280 in attendance, was held at the Severin Hotel in Indianapolis, January 9-11, D. D. Baker of Wabash presiding.

New officers for the year are R. E. Kepner of New Castle, president; G. E. Botkin of Marion, vice-president; and Charles C. Dobson of New Augusta, secretary-treasurer. J. E. Carrico of Bicknell and Harry Brown of Fort Wayne were elected to the Board of Directors, each for a term of three years. J. L. Axby and F. H. Brown of Indianapolis were chosen delegate and alternate, respectively, to the House of Representatives of the national association.

The Board of Directors recommended that the Association vigorously oppose section 5, article X of the proposed administrative by-laws of the A.V.M.A., which provides for the admittance of non-veterinarians to associate membership, with the same rights, status and privileges as active members. The recommendation of the Board was unanimously accepted and Delegate Axby was advised of the wishes of the Association in this matter.

At the annual banquet on January 10, the presentation of a portrait of the late R. A. Craig of Purdue University was made by J. L. Axby and accepted by Edward C. Elliott, president of the University. The Association sponsored the painting of the portrait, for which Dr. Craig posed, following a resolution unanimously approved at its 1939 meeting. Both Dr. Axby's address and Dr. Elliott's speech of acceptance were impressive tributes to Dr. Craig's fine character.

After the banquet, President Kepner presented, on behalf of the Association, a beautiful past-president's gold key to each of 20 former presidents.

The small animal clinic on the last day of the meeting, conducted by R. L. Tinkham of Chicago, concluded one of the most successful conventions ever staged by Indiana veterinarians.

CHARLES C. DOBSON, *Secretary.*

Iowa

At a meeting of the Botna Valley Veterinary Medical Association held at Atlantic, February 12, the following officers were elected: L. M. Getz of Atlantic, president; C. C. Lawrence of Manilla, vice-president; and R. S. Beaver of Harlan, secretary-treasurer.

State Veterinarian H. A. Seidell of Des Moines was the guest speaker.

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Sixty-nine veterinarians attended the Lincoln's birthday meeting of the Cedar Valley Veterinary Medical Association at Black's tea room in Waterloo, February 12.

Sound films, furnished by the Winthrop Chemical Co., were shown on artificial insemination in mares and cows and reproductive disorders in horses and cattle. C. H. Covault of Iowa State College, Ames, spoke on enzoötic calf pneumonia, infectious diarrhea of cows, ketosis, and sweet clover poisoning. He told of the successful use of sulfapyridine for calf pneumonia. The dosage he recommended is 5 Gm. (77 gr.) for a 27-kg. (60-lb.) calf every six hours during the first 24 hours. For the nervous form of ketosis, Dr. Covault recommended the use of 30-Gm. (1-oz.) doses of chloral hydrate with sodium bicarbonate, and green grass if practicable.

A resolution was passed congratulating the Associated Serum Producers on the excellent publicity they are sponsoring in behalf of the veterinary profession.

The question box, always a popular feature of Cedar Valley meetings, was conducted by President Guy C. Brown of Hudson.

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Despite adverse weather and highway conditions, 36 veterinarians attended the dinner meeting of the East Central Veterinary Medical Society at the Hotel Jefferson in Iowa City, February 8. Both a literary and an epicurean treat were provided by the Johnson County Veterinary Medical Society, which sponsored the affair.

Among the contributors to the program were Lee Cochran of the University of Iowa, Iowa City; Chemist Philip W. West of Iowa City; Inspector-in-Charge J. A. Barger of the federal bureau of animal industry, Des Moines; I. W.

Moranville of Durant, president of the Eastern Iowa Veterinary Association, Inc.; Lou Nelson of Ames; R. M. Hofferd of Cedar Rapids; and S. B. Watson of Washington.

Timothy P. White, former representative of the U. S. bureau of animal industry in London, England, and recently transferred to Rockford, Ill., gave a highly informative talk on European veterinarians, their customs, colleges, literary societies, disease-control measures and laws, and meat-inspection service. Dr. White praised the meat-inspection service of the United States Department of Agriculture as the best in the world and classed that service in Poland, before the present war, as rating next to ours in scope and efficiency. He also pointed out the tremendous difference in the sanitation in American packing plants as compared with that in European abattoirs.

JAMES W. PIRIE, *Secretary.*

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A notable event in Iowa veterinary history is the retirement of C. J. Scott of Knoxville as secretary of the Iowa Veterinary Medical Association. Ill health and heavy demands on his time from tasks outside of the secretaryship necessitated his abandoning the office. C. C. Franks of Grimes was elected to succeed him at the annual meeting of the Association, held in Des Moines, January 23-25. Dr. Scott was presented with a luxurious office chair and extended a life membership in the Association.

Guy S. Jones of Audubon is president of the Iowa organization and G. G. Baker of Spencer, president-elect.

A. H. QUIN, *Resident Secretary.*

Kansas

Sixteen veterinarians, representatives of the various phases of veterinary activity in the state, met recently in Wichita as guests of J. A. Bogue for a frank, "down to earth" discussion of current veterinary problems. It was agreed that there should be more of these informal gatherings, that they would help veterinarians to serve their clients more efficiently.

Manitoba

The 50th annual meeting of the Veterinary Association of Manitoba was held in the Marlborough Hotel, Winnipeg, on February 9, J. A. Allen of Winnipeg presiding. More than 40 veterinarians were in attendance, many of whom are practitioners from country points.

Officers for the ensuing year were elected as follows: D. J. Lawson of Shoal Lake, president; O. McGuirk of Dauphin, vice-president; Wm. Hilton of Winnipeg, secretary-treasurer and registrar. A. Savage of Winnipeg, H. H. Ross of Brandon, J. A. Allen, and R. H. Lay of Winnipeg were elected to the council.

Major W. Shearer spoke on "The Army Veterinary Corps," tracing its development from the early days of its organization in England to the present time.

A symposium on swine disorders followed, which comprised the following papers: "Nutrition and Nutritional Disorders of the Pig," by G. W. Wood of the University of Manitoba; "Hog Cholera," by R. H. Lay of the Dominion department of agriculture; "Hemorrhagic Septicemia of Swine," by J. M. Isa of the Provincial Pathological Laboratory, Winnipeg; "Hog Diseases from a Practitioner's Viewpoint," by J. A. Martin of Sperling; and "Swine Erysipelas," by A. Savage, provincial animal pathologist, Winnipeg.

Concluding the literary program, J. A. Allen, pathologist in the game and fisheries branch of the Manitoba government, presented an illustrated address, entitled "The Veterinarian and Mink Diseases."

An anniversary banquet and program were held in the evening, with the veterinarians and their wives and a number of guests attending. Alfred Savage was the master of ceremonies. Special tribute was paid to the pioneer members of the Association, many of whom were present.

Massachusetts

Edgar A. Crossman will assume the office of dean of the veterinary school at Middlesex University in June, when he retires from the federal bureau of animal industry after 40 years of service.

Born in Vanceboro, Maine, on May 21, 1870, Dr. Crossman received the degree of Doctor of Veterinary Medicine from Harvard University in 1891. He entered the service of the B.A.I. in 1900 and, in 1918, was appointed inspector in charge of tuberculosis eradication for New England. In 1928, he became a senior veterinarian.

Dr. Crossman is president of the Eastern States Live Stock Loss Prevention Board, past president of the Massachusetts Veterinary Association, past president of the National Association of Bureau of Animal Industry Veterinarians, past president of the Boston section of the United States Department of Agriculture Club, first vice-president of the United States Live Stock Sanitary Association, and a member of the A.V.M.A., New Hampshire Veterinary Association, New England Veterinary Medical Association, and Boston Medical Milk Commission.

He was formerly chairman of the Needham (Mass.) board of health and is a past president of the Needham board of trade.

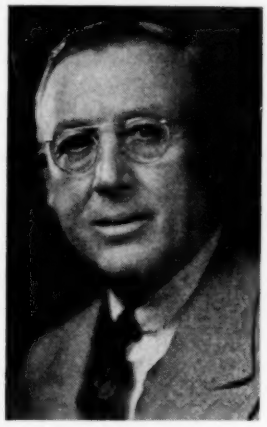
Michigan

Three prominent veterinarians—E. Jungherr of Storrs, Conn.; A. J. Durant of Columbia, Mo.; and C. D. Lee of Ames, Iowa—have been selected to serve on a committee which will act

in an advisory capacity in connection with the activities of the Regional Poultry Research Laboratory at East Lansing. Neurolymphomatosis is the disease being given the greatest amount of the laboratory's attention at the present time. J. Holmes Martin is director of the laboratory and C. A. Brandly is senior pathologist.

Minnesota

The board of regents of the University of Minnesota has elected W. L. Boyd chief of the division of veterinary medicine in the University's department of agriculture at University Farm, St. Paul, to fill the post left vacant by the death of C. P. Fitch.



W. L. Boyd

Dr. Boyd joined the staff of the division of veterinary medicine as an instructor in 1911. In 1912 he became an assistant professor and, in 1918, he was awarded a professorship in the division. His first year with the University was given over largely to the production of anti-hog-cholera serum and hog-cholera virus. In 1912, he was placed in charge of the clinical services to the herds and flocks at the experiment station. At that time he became profoundly interested in veterinary gynecology and the clinical and pathological phases of Bang's disease and its sequels. He has contributed many important papers pertaining to this field during the past two decades.

He is a member of the American Veterinary Medical Association and the Minnesota State Veterinary Medical Society, the Minnesota Academy of Science, the Research Workers in Animal Diseases in North America, the Minnesota Stallion Registration Board, the athletic board of the University, and a past member and chairman of the state veterinary examining board. He is also a member of Sigma Xi, honorary scientific society, and Gamma Sigma Delta, honor society in agriculture.

New Jersey

The 56th annual meeting of the Veterinary Medical Association of New Jersey was held at the Hotel Douglas, Newark, on January 16-17, with 140 members and guests in attendance.

During the convention the following papers were presented:

The Diagnosis of Chronic Mastitis—James H. Murphy, Beemerville.

The Occurrence of Eastern Strain Encephalomyelitis Virus in New Jersey Pheasantries—R. A. Hendershott, Trenton.

Simplified Veterinary Radiography—Raymond J. Garbutt, New York, N. Y.

Artificial Insemination in Mares and Cows (motion picture demonstration)—Charles E. Fanslau, New York, N. Y.

Cystic Ovaries in Bovines and Their Treatment—Robert P. Lawrence, Verona.

Cysts—Howard J. Milks, Ithaca, N. Y.

Poultry Diseases and Their Economic Importance—Henry Van Roekel, Amherst, Mass.

Worm-Host Systems as Labile Mechanisms: A View of the Nematode Ruminant Problem—Norman R. Stoll, Princeton.

Acetonemia in the Dairy Cow—Roger S. Amadon, Philadelphia, Pa.

Two "dry clinic" sessions were held during the two-day meeting, the following discussions and case reports being presented:

Coöperative Dog-Food-Testing Program of the A.V.M.A.-A.A.H.A.—M. L. Morris, Stelton.

New Methods of Fixation for Double Luxation of the Hip—John D. Devine, South Orange.

Encephalitis in Dogs of Fox Origin—Armour C. Wood, Trenton.

Hernia and Dystokia Complications—Arthur F. North, Jr., Somerville.

Sulfanilamide—W. R. Ecker, Newark.

Pyometra—J. B. Engle, Summit.

A Case of Blackleg—H. W. Peele, Woodstown.

Infectious Enteritis of Dogs—E. R. Cushing, Plainfield.

The banquet on the evening of January 16 was attended by more than 100 members, their wives and guests. Willard H. Allen, state secretary of agriculture, and A. E. Wight, president-elect of the national association, were the guest speakers.

Officers elected for the ensuing year are G. H. Kimnach of Highstown, president (reëlected); J. T. McGrann of Trenton, first vice-president; R. A. Hendershott of Trenton, second vice-president; J. G. Hardenbergh of Plainsboro, secretary (reëlected); and J. B. Engle of Summit, treasurer (reëlected).

J. G. HARDENBERGH, *Secretary.*

New York

Cornell University's annual conference for veterinarians was held on January 10-12 at the New York State Veterinary College, Cornell University, Ithaca, with a total of 325 veterinarians in attendance. The first day was devoted to large animal practice, the second to general practice, and the last day to small animal diseases. Some of the featured topics were rabies, equine encephalomyelitis, and calfhood vaccination for Bang's disease.

The following papers were presented:

A Report of Some Interesting Cases from the Surgical Clinic—A. G. Danks, Ithaca.

The Anterior Pituitary-Lobe Hormones in the Treatment of Ketosis in the Dairy Cow—M. G. Fincher and C. E. Hayden, Ithaca.

Gastrointestinal Parasites of Sheep and Their Control—D. W. Baker, Ithaca (in absence of R. E. Rebrassier, Columbus, Ohio).

The Professions and the Public—Milton E. Loomis, Albany.

The Epizoötiology of Infectious Equine Encephalomyelitis with Special Reference to Its Diagnosis—L. T. Giltner, Washington, D. C.

Prophylaxis in Equine Encephalomyelitis—R. A. Kelser, Washington, D. C.

The Parasitic Hazards Encountered in Southern and Western Raised Calves and Lambs in New York—D. W. Baker, Ithaca.

A Practitioner's Experience in the Diagnosis and Control of Parasitic Gastroenteritis in Imported Steers—Harry R. Greene, Brockport.

Listerella Infection (Circling Disease) of Sheep, Goats and Cattle—Peter Olafson, Ithaca.

Measures for the Control and Prevention of Rabies—R. A. Kelser.

Progress Report on a Vaccination Experiment for Bang's Disease—R. R. Birch, Ithaca.

X-Ray Therapy in Veterinary Medicine—M. A. Emmerson, Philadelphia, Pa.

The Cesarean Operation—H. C. Stephenson, Ithaca.

The Principles Underlying the Use of Biologics—A. Zeissig, Ithaca.

Sex Determination—H. T. Batt, Ithaca.

Methods of Diagnosis and Treatment of Internal Parasitisms of Small Animals—D. W. Baker (in the absence of R. E. Rebrassier).

Ticks and Disease with Special Reference to Spotted Fever in the Eastern States—R. E. Matheson, Ithaca.

Neoplasms in Dogs—R. B. McClelland, Buffalo.

Canine Filariasis—Walter E. Hobbs, Columbus, Ohio.

Diseases of the Digestive Apparatus—C. P. Zepp, New York.

Cysts—Howard J. Milks, Ithaca.

The evening of the first day the Cornell Veterinary Alumni Association held its annual dinner and meeting on the campus, with approximately 200 in attendance. The second afternoon a special program in laboratory and clinical practice was given in which each department of the College participated.

The conference dinner held on the second evening made a new attendance record. Two hundred and seventy-five were present and heard Dean W. A. Hagan extend the greetings of the College. Talks on "The 1939 Football Season," by Walter Matuszczak, veterinary student and captain-elect for 1940; "Reporting and Interpreting War News," by Harry Stutz; and "Public Hospitality Around the World," by Howard B. Meek, completed the program.

Charles E. Fanslau of the Winthrop Chemical Co. showed several films during the conference, and exhibits were prepared by W. C. Muenscher on poisonous plants, Robert E. Matheson on ticks, and Donald Cameron on fleas.

The extension of the program from the usual two days to three proved highly successful.

W. S. STONE, *Reporter.*

Oklahoma

Oklahoma needs several more graduate veterinarians, and there are a number of county-seat towns in which a good practice could be built in a short time.

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The 25th annual meeting of the Oklahoma Veterinary Medical Association was held at Oklahoma City, January 8-9, with headquarters at the Skirvin Hotel. The meeting was a success in spite of the fact that snow-bound roads kept many away.

The following papers were presented during the two-day session:

Fungus Infections of the Skin of Dogs and Cats—H. C. Smith, Fort Dodge, Iowa.

Viruses—Frank Breed, Lincoln, Neb.

Mineral Deficiencies — Hubert Schmidt, College Station, Texas.

Problems of Small Animal Practice—J. C. Flynn, Kansas City, Mo.

Plastic Surgery of Small Animals—R. L. Anderes, Kansas City, Mo.

Foci Infection in Canine Pathology—H. C. Smith.

Rabies—J. C. Flynn.

Report of Delegate to the House of Representatives—W. F. Irwin, Tulsa.

Artificial Insemination (motion pictures)—R. L. Anderes.

The proposed code of ethics of the national association was read and discussed, and on motion was laid over for the summer meeting, in order that the members may have time to study it.

Over 90 veterinarians and their wives attended the banquet and dance held in the Venetian room of the Skirvin on the evening of the first day. Frank Breed acted as toastmaster.

An election of officers resulted as follows: C. H. McElroy of Stillwater, president; W. F. Irwin of Tulsa, vice-president; and F. Y. S. Moore of McAlester, secretary-treasurer (reëlected).

F. Y. S. MOORE, *Secretary.*

Oregon

The Willamette Valley Veterinary Medical Association met at Woodburn on January 10 and elected the following officers for the forthcoming year: Geo. F. Reid of Albany, president; Fay Rankin of Clatskanie, vice-president; and T. Robert Phelps of Oregon City; secretary-treasurer (reëlected).

A resolution was passed to support C. H. Seagraves for district 7 of the national association's Executive Board. A committee consisting of F. W. Lange of Salem, W. B. Coon of Forest Grove, and I. C. Robison of McMinnville was appointed to make up a suggested schedule of fees for the information of new veterinarians.

South Carolina

The South Carolina Association of Veterinarians held their annual business meeting on January 23 at the Jefferson Hotel, Columbia, with about 40 in attendance. Routine matters were discussed and plans were made for the summer meeting, to be held at Greenville.

South Dakota

The South Dakota Veterinary Medical Association's 19th annual meeting was held at the Hotel Carpenter in Sioux Falls, December 14-15, 1939.

On the morning of the 14th, a clinical program was presented at the Sioux Falls sales pavilion. The afternoon session was opened

Photos from the

Journal's Candid Camera

MISSOURI VETERINARIANS

Top left (left to right): C. R. Kinnison, Chillicothe; C. A. Schulz (standing), Independence; M. S. Campbell, Brookfield; and R. S. Shipp, Cameron. Top right (left to right): H. E. Curry, Jefferson City; T. E. Wilke, West Plains; Clarence L. Campbell, St. Louis; J. C. Flynn, Kansas City; and W. H. Bailey, St. Louis.

Center above (left to right): John L. Wells, Kansas City, Mo.; Kirtley Sears, Maryville; Claude T. Old, Sikeston; E. T. Hallman, East Lansing, Mich.; S. W. Haigler, St. Louis; and E. K. Sales, East Lansing, Mich. (All of the above pictures and that shown at the right were taken at the annual meeting of the Missouri Veterinary Medical Association, Columbia, Mo., January 31 to February 2, 1940.)

Right (left to right): D. F. Luckey, St. Louis; S. N. Smith, Columbia; R. C. Dickson, Kansas City; J. A. Thurman, Eolia.

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Bottom left (standing, left to right): Evar Cedarleaf and John Salsbury; (seated, left to right) Donn Becon and Max Benson—all staff members of The Veterinary Student, official publication of the Iowa State College Student Chapter of the A.V.M.A. Bottom right (left to right): Iowa Veterinarians George B. Senior of Creston, C. C. Franks of Grimes, and James W. Pirie of Cedar Rapids.

with an address of welcome by John McKee, mayor of Sioux Falls. The technical program for the afternoon included a talk and slide demonstration on swine diseases by H. C. H. Kernkamp of the University of Minnesota, St. Paul, Minn. "Effects of Heat on Phenolized Virus" was presented by J. D. Ray of Omaha, Neb. H. J. Classick of Sioux Falls spoke on the progress made in Bang's disease control in South Dakota.

On the evening of the first day, about 100 veterinarians and their wives and guests sat down to a fine dinner followed by several interesting talks by out-of-state veterinarians.

Contributors to the literary program of the second day were E. J. Frick of Kansas State College, Manhattan, Kan., who presented a paper on cattle diseases; H. E. Schwermann of New Ulm, Minn., who spoke on the selling of veterinary services; and L. M. Roderick of Kansas State College, who gave a paper on sheep diseases.

The following men were elected to office for the ensuing year: J. M. Baily of Parker, president; E. M. Walker of Brookings, vice-president; and George E. Melody of Gettysburg, secretary-treasurer (reëlected).

GEORGE E. MELODY, *Secretary.*

Tennessee

A. C. Topmiller, state veterinarian, reports 17 outbreaks of hog cholera in ten counties of the state during January, seven outbreaks of erysipelas in six counties, five of hemorrhagic septicemia in four counties, one of blackleg and one of rabies.

Texas

A regular monthly meeting of the Houston Veterinary Association was held at Frank Hecker's office on the evening of February 1.

J. Gilbert Horning read a paper on typhus and its relation to the veterinarian which was ably supplemented by J. J. Reid, who related his experience when suffering some time ago with this disease. A round-table discussion on heartworms in dogs was led by Madero N. Bader. A questionnaire prepared by W. T. Hufnall and J. Gilbert Horning resulted in Dr. Bader's winning first prize and John T. Kirby, second prize.

J. GILBERT HORNING, *Corres. Sec'y.*

Vermont

The Vermont Veterinary Medical Association held its annual meeting at the Hotel Vermont in Burlington, January 13, E. K. Treat of Manchester Center presiding.

J. J. Staab of Montpelier was elected president for the ensuing year; A. A. Mortimer of Randolph, 1st vice-president; A. D. Spooner of Barre, 2nd vice-president; and G. N. Welch of Northfield, secretary-treasurer (reëlected).

Dr. Staab was chosen as delegate to the A.V.M.A. House of Representatives with the power to appoint his alternate.

Dinner followed by a program of entertainment concluded the meeting.

G. N. WELCH, *Secretary.*

Virginia

A sum of $50 has been appropriated by the Virginia State Veterinary Medical Association toward the entertainment of members of the national association during the latter's meeting at Washington, D. C., in August.

Wisconsin

The Central Wisconsin Veterinary Medical Association held a successful clinic and literary program at Eau Claire, December 13. Geo. B. Wrigglesworth entertained the group at his hospital, where the clinic was held. Sixty members were in attendance.

COMING MEETINGS

Small Animal Hospital Association. Los Angeles, Calif. March 5, 1940. R. W. Gerry, secretary, 8474 Melrose Ave., Los Angeles, Calif.

New York City, Veterinary Medical Association of. Hotel New Yorker, New York, N. Y. March 6, 1940. J. J. Merenda, secretary, 136 W. 53rd St., New York, N. Y.

Dallas-Fort Worth Veterinary Medical Society. Fort Worth, Texas. March 7, 1940. H. V. Cardona, secretary, 2736 Purington Ave., Fort Worth, Texas.

Houston Veterinary Association. Houston, Texas. March 7, 1940. W. T. Hufnall, secretary, 1612-14 E. Alabama Ave., Houston, Texas.

Midwest Small Animal Association. Hotel Burlington, Burlington, Iowa. March 7, 1940. C. L. McGinnis, secretary, 1314 Main St., Peoria, Ill.

Ak-Sar-Ben Veterinary Medical Association. Fontenelle Hotel, Omaha, Neb. March 11, 1940. J. D. Ray, secretary, 1124 Harney St., Omaha, Neb.

Chicago Veterinary Medical Association. Hotel Sherman, Chicago, Ill. March 12, 1940. G. S. Elwood, secretary, 5449 Broadway, Chicago, Ill.

Southeastern Michigan Veterinary Medical Association. Medical Arts Bldg., 3919 John R St., Detroit, Mich. March 13, 1940. F. D. Egan, secretary, 17422 Woodward Ave., Detroit, Mich.

St. Louis District Veterinary Medical Association. Melbourne Hotel, St. Louis, Mo. March

13, 1940. J. P. Torrey, secretary, 610 Veronica Ave., East St. Louis, Ill.

Willamette Valley Veterinary Medical Association. Albany, Ore. March 13, 1940. T. Robert Phelps, secretary, 1514 Washington St., Oregon City, Ore.

American Foundation of Veterinary Therapy. Hotel President, Kansas City, Mo. J. C. Flynn, 3026 Main St., Kansas City, Mo.

Kansas City Veterinary Medical Association. Kansas City, Mo. March 18, 1940. S. J. Schilling, secretary, Box 167, Kansas City, Mo.

San Diego County Veterinary Medical Association. Zoölogical Research Bldg., Balboa Park, San Diego, Calif. March 18, 1940. Paul D. DeLay, secretary, State Poultry Pathological Laboratory, Balboa Park, San Diego, Calif.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. March 20, 1940. Charles Eastman, secretary, 725 S. Vancouver Ave., Los Angeles, Calif.

Alabama Veterinary Medical Association and Conference for Graduate Veterinarians. School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala. March 20-23, 1940. I. S. McAdory, dean, School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala.

Bexar County, The Veterinary Medical Association of. Gunter Hotel, San Antonio, Texas. March 21, 1940. W. A. Lawrence, secretary, 3231 W. French Pl., San Antonio, Texas.

Long Island Veterinary Medical Association. Long Island, N. Y. March 21, 1940. Herman Tax, secretary, State Institute, Farmingdale, Long Island, N. Y.

Arkansas Veterinary Medical Association. Hot Springs, Ark. March 21-22, 1940. F. O. Garrett, secretary, Dumas, Ark.

Keystone Veterinary Medical Association. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. March 27, 1940. A. Henry Craige, Jr., secretary, University of Pennsylvania, Philadelphia, Pa.

Massachusetts Veterinary Association. Hotel Westminster, Copley Square, Boston, Mass. March 27, 1940. H. W. Jakeman, secretary, 44 Bromfield St., Boston, Mass.

American Animal Hospital Association. Hollywood-Roosevelt Hotel, Hollywood, Calif. March 27-30, 1940. D. A. Eastman, secretary, 2635 N. W. 36th St., Miami, Fla.

American Scientific Congress. Washington, D. C. May 10-16, 1940. (A. V. M. A. special committee to represent veterinary profession of United States: Col. R. A. Kelser, John R. Mohler and Willard H. Wright.)

American Veterinary Medical Association. Mayflower Hotel, Washington, D. C. August 26-30, 1940. L. A. Merillat, secretary, 221 N. La Salle St., Chicago, Ill.

PERSONAL NOTES

Births

To Dr. (Iowa '39) and Mrs. A. L. McGrath of Jesup, Iowa, a daughter, Miriam Lorene, September 8, 1939.

To Dr. (Colo. '35) and Mrs. Vernon V. Golden of Omaha, Neb., a son, Stewart Vernon, November 14, 1939.

To Lt. (Colo. '34) and Mrs. Karl H. Willers of Fort Oglethorpe, Ga., a son, Karl Robert, November 11, 1939.

Marriage

E. L. Kelley (Tex. '39) of Utopia, Texas, to Yvonne McGarry of Carlsbad, N. Mex., January 1, 1940.

Activities

Emmet W. Paul (Wash. '37) recently left CCC duty at Fort Douglas, Utah, and opened the Redwood Veterinary Hospital at Redwood City, Calif.

Karl L. Sutton (Mich. '36) has resigned from the service of the federal bureau of animal industry and is now engaged in practice at Morenci, Mich.

Harry Brown (Ind. '23) of Fort Wayne, Ind., suffered a broken collar bone and a fractured wrist when he fell from a horse during a field trial on November 8.

C. L. Everson (O.S.U. '29) of the Maryland Live Stock Sanitary Service is on a leave of absence in Texas, where he is recovering from an acute attack of undulant fever.

H. Busman (Ont. '95), one-time inspector-in-charge of large B.A.I. forces in Chicago, Ill., and Indianapolis, Ind., was retired in October of 1939 and has taken up residence in Holland, Mich.

M. S. Arlein (Iowa '38) recently resigned from the service of the federal bureau of animal industry to accept a position as pathologist with the Angell Memorial Animal Hospital at Boston, Mass.

Leslie C. Murphy (Wash. '39), formerly of the Western Washington Experiment Station at Puyallup, Wash., has accepted a position as research veterinarian for the Carnation Milk Co. in Milwaukee, Wis.

Richard M. Scott (McK. '17) has sold his hospital in Los Angeles, Calif., to Albert A. Taylor (Wash. '37) and returned to Canada,

his native country, after almost 40 years of residence in the United States.

L. R. Haubrich (U. P. '11) and his son, Wilson R. (U. P. '37) of Claremont, N. H., are in charge of artificial insemination work for a dairymen's association of Sullivan county, N. H., and Windsor county, Vt.

Wallace V. Hornbacker (Mich. '38) has resigned from the service of the federal bureau of animal industry and is now engaged in artificial insemination work, supervised by the University of Wisconsin, at Oconto Falls, Wis.

L. C. Schantz (Corn. '30) of Albany, Ga., was seriously injured in an airplane crash at Butler, Ga., on September 26, 1939. His legs and jaw bones were broken and for a time it was not certain that he would recover. The latest report, however, was favorable.

Gustave A. Kay (Chi. '02) was recently retired from the meat-inspection force of the federal bureau of animal industry and has accepted a position with the Montana Livestock Sanitary Board at Missoula, Mont., as deputy state veterinarian in charge of state meat inspection.

DEATHS

E. J. Fallon of San Francisco, Calif., died on November 5, 1939.

A graduate of the San Francisco Veterinary College, class of 1908, Dr. Fallon joined the A.V.M.A. in 1911.

Andrew D. Ryan of Stewart, Minn. died on January 12, 1940.

Born in 1876, Dr. Ryan was graduated from the St. Joseph Veterinary College in 1910. He joined the A.V.M.A. in 1928.

L. J. Richards, 69, of Delaware, Ohio, died on October 16, 1939.

Dr. Richards held a V.S. degree (Ont. '03) and an M.D.V. degree (McK. '09). He joined the national association in 1916.

Frank W. Morgan, for 30 years a practitioner in Chattanooga, Tenn., died in Cocoa, Fla., of a heart attack on December 24, 1939.

A graduate of McKillip Veterinary College, class of 1906, Dr. Morgan joined the national association in 1906.

C. W. Boone of Roanoke, Va., was killed in an automobile accident on December 23, 1939.

Born at Boone Mill, Va., February 23, 1886, Dr. Boone was graduated from the Cincinnati Veterinary College in 1918. He joined the A.V.M.A. in 1937.

A. D. Gemmill of Celina, Ohio, died after a brief illness on November 7, 1939.

A graduate of the Ontario Veterinary College, class of 1892, Dr. Gemmill was in practice for 46 years in Celina. He joined the national association in 1911.

B. R. Parker, 72, died of a heart attack on January 11, 1940.

Born at Hebron, Wis., on March 23, 1867, Dr. Parker was graduated with highest honors from the Grand Rapids Veterinary College in 1901. He joined the national association in 1928.

H. Keown of Victoria, British Columbia, died on November 22, 1939.

Born at London, Ontario, March 27, 1876, Dr. Keown was graduated from the Ontario Veterinary College in 1907. He was in practice at Victoria for over 30 years.

Dr. Keown joined the national association in 1925.

Philip H. Radford, 48, of Slayton, Minn., died in January 1940.

Born at Swanwick, England, on April 15, 1891, Dr. Radford came to America while in his early teens. He was graduated from the Kansas City Veterinary College in 1918 and engaged in general practice thereafter. He joined the national association in 1919.

C. B. Griffiths, 44, supervising veterinary livestock inspector of the California state department of agriculture, died at his home in Sacramento, Calif., on January 16, 1940, following an illness of several months.

Born at Baileyville, Kan., April 13, 1895, Dr. Griffiths attended the Kansas State College, from which he was graduated in 1918. He was a member of the California State Veterinary Medical Association and the A.V.M.A., having joined the latter in 1918.

Among his survivors is a son, Clayton B., Jr., who is now in his first year at the Division of Veterinary Medicine, Kansas State College.

William Ross Sanderson, 52, died at his home in Brownwood, Texas, on January 14, 1940, after having been in ill health and confined to his bed since March of 1939.

Born in Lime Stone county, Ala., on July 5, 1887, Dr. Sanderson attended the Kansas City Veterinary College, from which he was graduated in 1915. He served in the federal bureau of animal industry for four years after his graduation, following which he entered private practice in Brownwood. He reëntered the bureau in July 1934 and served until January 1936, when he returned to his practice.

Dr. Sanderson was a member for ten years of the Texas State Board of Veterinary Examiners, and held the offices of president, vice-president and secretary of the Texas state association. He joined the national association in 1938.

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